This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

Digitized by Google

Digitized by Google

Digitized by Google

Digitized by Google

Digitized by Google

Digitized by Google

TRANSACTIONS OF THE

SECTION ON

Nervous and Mental Diseases

of the
American Medical Association
at the Seventieth Annual
Session, held at Atlantic City,
N. J., June 9 to 13, 1919

AMERICAN MEDICAL ASSOCIATION PRESS
CHICAGO: NINETEEN HUNDRED AND NINETEEN

Digitized by Google

BOSTON MEDICAL
SEP 24 1926
LIBRARY

CATALOGUED
SEP 24 1926
D. B. S.

Reprinted from
THE JOURNAL
of the
AMERICAN MEDICAL ASSOCIATION

Copyright, 1919
by the
American Medical Association

Digitized by Google

OFFICERS FOR 1918-1919

CHAIRMAN

ARCHIBALD CHURCH - - - - - - - - - - - - - CHICAGO

VICE CHAIRMAN

E. E. SOUTHARD - - - - - - - - - - - - - - BOSTON

SECRETARY

CHARLES W. HITCHCOCK - - - - - - - - - - DETROIT

EXECUTIVE COMMITTEE

F. X. DERCUM, PHILADELPHIA
G. A. MOLEEN, DENVER
CHARLES EUGENE RIGGS, ST. PAUL

OFFICERS FOR 1919-1920

CHAIRMAN

ELMER E. SOUTHARD - - - - - - - - - - - - - BOSTON

VICE CHAIRMAN

ARTHUR S. HAMILTON - - - - - - - - - - MINNEAPOLIS

SECRETARY

CHARLES W. HITCHCOCK - - - - - - - - - - - DETROIT

EXECUTIVE COMMITTEE

G. A. MOLEEN, DENVER
BERNARD SACHS, NEW YORK
ARCHIBALD CHURCH, CHICAGO

Digitized by Google

Digitized by Google

LIST OF OFFICERS

This Section has been in existence under its present name since 1900. The branches of the practice of medicine which comprise its province were classified with some other subjects in the early days, as is shown by the following section names. The list of officers has been compiled from the published records, which are not complete—for example, the city is not given in some cases. It will be appreciated if any additional information is brought to the attention of the Secretary of the American Medical Association.

SECTION ON PSYCHOLOGY

1866 Chairman, C. A. Walker, Boston.

1867 Chairman, C. A. Lee, Peekskill, N. Y.
Secretary, H. R. Stover, Boston.

1868 Chairman, D. F. Condie, Philadelphia.
Secretary, C. A. Lee, Peekskill, N. Y.

1869 Records wanting.

1870 Chairman, J. P. Gray, Utica, N. Y.
Secretary, A. Hartmann, Baltimore.

1871 Records wanting.

1872 Chairman, Isaac Ray, Philadelphia.
Secretary, J. Curwen, Harrisburg, Pa.

SECTION ON MEDICAL JURISPRUDENCE, CHEMISTRY AND PSYCHOLOGY

1873 Chairman, A. N. Talley, Columbia, S. C.
Secretary, E. L. Howard, Baltimore.

1874 Chairman, Jerome Cochran, Montgomery, Ala.
Secretary, G. A. Moses, St. Louis.

1875 Chairman, E. L. Howard, Baltimore.
Secretary, E. A. Hildreth, Wheeling, W. Va.

1876—Chairman, Eugene Grissom, Raleigh, N. C.
Secretary, E. A. Hildreth, Wheeling, W. Va.

1877 Chairman, W. Kempster, Oshkosh, Wis.
Secretary, E. A. Hildreth, Wheeling, W. Va.

1878 Chairman, W. M. Compton, Holly Springs, Miss.
Secretary, L. M. Eastman, Baltimore.

SECTION ON STATE MEDICINE, PUBLIC HYGIENE, CHEMISTRY AND PSYCHOLOGY

1879 Chairman, J. F. Hibberd, Richmond, Ind.
Secretary, T. F. Wood, Wilmington, N. C.

SECTION ON STATE MEDICINE

1880 Chairman, J. T. Reeve, Appleton, Wis.
Secretary, R. G. Jennings, Little Rock, Ark.

1881 Chairman, A. L. Gibson, U. S. Navy.
Secretary, J. H. Sears, Waco, Texas.

1882 Chairman, Foster Pratt, Kalamazoo, Mich.
Secretary, Thomas L. Neal, Dayton, Ohio.

1883 Chairman, D. J. Roberts, Waterville, Tenn.
Secretary, C. W. Franzoni, Washington, D. C.

1884 Chairman, E. W. Schaufler, Kansas City, Mo.
Secretary, J. N. McCormack, Bowling Green, Ky.

Digitized by Google

LIST OF OFFICERS—Continued

1885 Chairman, J. H. Rauch, Chicago.
Secretary, F. E. Daniell, Austin, Texas.

1886 Chairman, G. H. Rohe, Baltimore.
Secretary, W. Wyman, Washington, D. C.

1887 Chairman, H. B. Baker, Dighton, Mich.
Secretary, S. T. Armstrong, U. S. M.-H. Service.

1888 Chairman, J. B. Lindsley, Nashville, Tenn.
Secretary, S. T. Armstrong, U. S. M.-H. Service.

1889 Chairman, J. B. Hamilton, Washington, D. C.
Secretary, F. S. Bascom, Salt Lake City.

1890 Chairman, J. D. Plunkett, Nashville, Tenn.
Secretary, F. S. Bascom, Salt Lake City.

1891 Chairman, Benjamin Lee, Philadelphia.
Secretary, L. F. Flick, Philadelphia.

1892 Chairman, C. A. Lindsley, New Haven, Conn.
Secretary, S. P. Duffield, Detroit.

1893 Chairman, G. W. Stoner, U. S. M.-H. Service.
Secretary, C. H. Shepard, Brooklyn.

1894 Chairman, L. H. Montgomery, Chicago.
Secretary, C. H. Shepard, Brooklyn.

1895 Chairman, C. H. Shepard, Brooklyn.
Secretary, Elmer Lee, Chicago.

1896 Chairman, Elmer Lee, Chicago.
Secretary, L. F. Bishop, Morristown, N. J.

1897 Chairman, I. N. Quimby, Jersey City, N. J.
Secretary, A. R. Reynolds, Chicago.

1898 Chairman, A. R. Reynolds, Chicago.
Secretary, W. P. Munn, Denver.

1899 Chairman, W. C. Woodward, Washington, D. C.
Secretary, Amand Ravold, St. Louis.

SECTION ON MEDICAL JURISPRUDENCE

1887 Chairman, F. M. Reid, Baltimore.
Secretary, C. B. Belt, Boston.

1888 Chairman, J. G. Kiernan, Chicago.
Secretary, T. B. Evans, Baltimore.

1889 Chairman, T. B. Evans, Baltimore.
Secretary, T. D. Crothers, Hartford, Conn.

1890 Chairman, T. D. Crothers, Hartford, Conn.
Secretary, H. N. Moyer, Chicago.

SECTION OF MEDICAL JURISPRUDENCE AND NEUROLOGY

1891 Chairman, H. N. Moyer, Chicago.
Secretary, G. D. Strawbridge, Philadelphia.

1892 Chairman, C. K. Mills, Philadelphia.
Secretary, J. G. Kiernan, Chicago.

1893 Chairman, J. G. Kiernan, Chicago.
Secretary, E. P. Norbury, Jacksonville, Ill.

1894 Chairman, D. R. Brower, Chicago.
Secretary, W. J. Gavigan, San Francisco.

1895 Chairman, T. D. Crothers, Hartford, Conn.
Secretary, W. J. Herdman, Ann Arbor, Mich.

1896 Chairman, W. J. Herdman, Ann Arbor, Mich.
Secretary, C. H. Hughes, St. Louis.

1897 Chairman, C. H. Hughes, St. Louis.
Secretary, H. T. Patrick, Chicago.

1898 Chairman, F. Peterson, New York.
Secretary, H. T. Patrick, Chicago.

1899 Chairman, H. T. Patrick, Chicago.
Secretary, F. S. Pearce, Philadelphia.

Digitized by Google

LIST OF OFFICERS—Continued

SECTION ON NERVOUS AND MENTAL DISEASES

1900 Chairman, H. A. Tomlinson, St. Peter, Minn.
Secretary, F. Savary Pearce, Philadelphia.

1901 Chairman, R. Dewey, Milwaukee, Wis.
Secretary, F. Savary Pearce, Philadelphia.

1902 Chairman, John E. Weeks, New York.
Secretary, Frank Todd, Minneapolis.
Delegate, Frank Allport, Chicago.

1903 Chairman, F. Savary Pearce, Philadelphia.
Secretary, David I. Wolfstein, Cincinnati.
Delegate, F. W. Langdon, Cincinnati.

1904 Chairman, J. H. McBride, Los Angeles.
Secretary, David I. Wolfstein, Cincinnati.

1905 Chairman, Wharton Sinkler, Philadelphia.
Secretary, David I. Wolfstein, Cincininati.
Delegate, J. H. McBride, Los Angeles.

1906 Chairman, Morton Prince, Boston.
Secretary, T. H. Weisenburg, Philadelphia.
Delegate, D'Orsay Hecht, Chicago.

1907 Chairman, T. H. Weisenburg, Philadelphia.
Secretary, William Alexander Jones, Minneapolis.
Delegate, Morton Prince, Boston.

1908 Chairman, M. Allen Starr, New York.
Secretary, William Alexander Jones, Minneapolis.
Delegate, T. H. Weisenburg, Philadelphia.

1909 Chairman, Theodore Diller, Pittsburgh.
Secretary, William Alexander Jones, Minneapolis.
Delegate, M. Allen Starr, New York.

1910 Chairman, W. A. Jones, Minneapolis.
Vice Chairman, Herman H. Hoppe, Cincinnati.
Secretary, E. E. Southard, Cambridge, Mass.

1911 Chairman, Edward D. Fisher, New York.
Vice Chairman, George H. Moody, San Antonio, Texas.
Secretary, E. E. Southard, Cambridge, Mass.
Delegate, W. A. Jones, Minneapolis.

1912 Chairman, H. T. Pershing, Denver.
Vice Chairman, W. W. Graves, St. Louis.
Secretary, G. A. Moleen, Denvxer.
Delegate, E. B. Angell, Rochester, N. Y.

1913 Chairman, W. W. Graves, St. Louis.
Vice Chairman, C. D. Camp, Ann Arbor, Mich.
Secretary, G. A. Moleen, Denver.
Delegate, G. Eugene Riggs, St. Paul.

1914 Chairman, F. X. Dercum, Philadelphia.
Vice Chairman, H. G. Brainerd, Los Angeles.
Secretary, G. A. Moleen, Denver.
Delegate, W. W. Graves, St. Louis.

1915 Chairman, George A. Moleen, Denver.
Vice Chairman, M. A. Bliss, St. Louis.
Secretary, A. S. Hamilton, Minneapolis.
Delegate, Milton B. Lennon, San Francisco.

1916 Chairman, Bernard Sachs, New York.
Vice Chairman, Charles W. Hitchcock, Detroit.
Secretary, A. S. Hamilton, Minneapolis.
Delegate, George A. Moleen, Denver.

1917 Chairman, Charles Eugene Riggs, St. Paul.
Vice Chairman, Charles W. Hitchcock, Detroit.
Secretary, A. S. Hamilton, Minneapolis.
Delegate, G. A. Moleen, Denver.

1918 Chairman, Archibald Church, Chicago.
Vice Chairman, E. E. Southard, Boston.
Secretary, Charles W. Hitchcock, Detroit.
Delegate, Hugh T. Patrick, Chicago.

1919 Chairman, Elmer E. Southard, Boston.
Vice Chairman, Arthur S. Hamilton, Minneapolis.
Secretary, Charles W. Hitchcock, Detroit.
Delegate, Hugh T. Patrick, Chicago.

Digitized by Google

Digitized by Google

CONTENTS

PAGE

List of Officers 7

Proceedings of the Section 13

Chairman's Address: The Neurology of Cervical Ribs. Archibald Church 17

Traumatic Neuroses in War and in Peace. T. H. Weisenburg 29

The Management of War Hysteria. Tom A. Williams .. 38

The Dual Personality of C. A. M. The Account of an Amnestic Fugue. Edward E. Mayer 59

Some Experiences in Neurologic Therapeutics. Charles K. Mills 85

The Range of the General Practitioner in Psychiatric Diagnosis. E. E. Southard 103

An Original Test for the Pathologic Great Toe Sign, with Illustrated Cases. Leo M. Crafts 116

A Case of Tumor of the Pons Varolii. Tom Bentley Throckmorton 123

Neurologic Sequelae Following Acute Respiratory Infections. William H. Robey, Jr., and Heiman Caro 127

Hysterical Hemiplegia. Report of Case Resulting from a Shrapnel Wound of the Scalp and Presenting Interesting Clinical Features. Harry H. Drysdale and J. S. S. Gardner 136

Congenital Tumor of the Brain (Telangiectasis) and Associated Cerebral Movements. William G. Spiller 149

A Contribution to the Histopathology of Epidemic ("Lethargic") Encephalitis. Peter Bassoe and George B. Hassin 158

The Rôle of the Pituitary Gland in Epilepsy. Beverly R. Tucker 169

The Resemblance of the Sensory Symptoms of Post-Diphtheritic Ataxia to Those Seen in the Cord Changes of Severe Anemia. George Wilson 177

A New Pluriglandular Compensatory Syndrome. Walter Timme 184

Index 205

Digitized by Google

Digitized by Google

PROCEEDINGS OF THE SECTION

Wednesday, June 11—Afternoon

The meeting was called to order at 2 o'clock by the chairman, Dr. Archibald Church, Chicago.

Dr. Archibald Church, Chicago, read the chairman's address, entitled "The Neurology of Cervical Ribs."

The chairman appointed the following members to act as a nominating committee: Drs. Sanger Brown, Chicago; Walter Timme, New York, and W. A. Jones, Minneapolis.

In the absence of two members of the executive committee made up of Drs. George A. Moleen, Denver, Bernard Sachs, New York, and C. Eugene Riggs, St. Paul, the chairman named Drs. T. H. Weisenburg, Philadelphia, and Hugh T. Patrick, Chicago, to act for Drs. Sachs and Riggs.

Dr. Joseph Byrne, New York, read a paper on "The Mechanism of Referred Pain, Hyperalgesia and Alcoholic Injections for the Relief of Neuralgia, with Suggestions for the Surgical Treatment of Injured Nerves." Discussed by Dr. Tom A. Williams, Washington, D. C.

Dr. Walter Timme, New York, read a paper on "A New Polyglandular Compensatory Syndrome." Discussed by Drs. Tom A. Williams, Washington, D. C., and Albert E. Sterne, Indianapolis.

Dr. Theodore H. Weisenburg, Philadelphia, read a paper on "Traumatic Neuroses on War and Peace."

Dr. Tom A. Williams, Washington, D. C., read a paper on "The Management of War Hysteria."

These two papers were discussed by Drs. Robert McGregor, Saginaw, Mich.; Charles A. Rosewater and W. W. Hicks, Newark, N. J.; Lewis J. Pollock, Chicago; T. H. Weisenburg, Philadelphia, and Tom A. Williams, Washington, D. C.

Dr. Edward E. Mayer, Pittsburgh, read a paper on "The Dual Personality of C. A. M.: The Account of an Amnesic Fugue." Discussed by Drs. Joseph Byrne, New York; H. D. Singer, Kankakee, Ill.; W. A. Jones, Minneapolis; Albert E. Sterne, Indianapolis; Tom A. Williams, Washington, D. C., and Edward E. Mayer, Pittsburgh.

Thursday, June 12—Morning

The following officers were elected: chairman, Dr. Elmer E. Southard, Boston; vice chairman, Dr. Arthur S. Hamil-

Digitized by Google

ton, Minneapolis; secretary, Dr. Charles W. Hitchcock, Detroit; delegate, Dr. Hugh T. Patrick, Chicago.

Dr. Hugh T. Patrick read the following resolution which was unanimously adopted:

WHEREAS, At the last session of the American Medical Association, the Section on Nervous and Mental Diseases petitioned the Trustees of the Association to establish a journal devoted to nervous and mental diseases and unanimously passed a resolution endorsing the plan; and

WHEREAS, The Trustees thereafter did establish the *Archives of Neurology and Psychiatry* and are successfully conducting the same; therefore, be it

Resolved, That this section hereby express to the Trustees of the American Medical Association and to the managing editor their appreciation of and thanks for this action.

Dr. Charles K. Mills, Philadelphia, read a paper on "Some Experience in Neurologic Therapeutics." No discussion.

Dr. Elmer E. Southard, Boston, read a paper on "The Range of the General Practitioner in Psychiatric Diagnosis." Discussed by Dr. E. Stanley Abbot, Belmont, Mass.

Dr. William G. Spiller, Philadelphia, read a paper on "Congenital Tumor of the Brain (Telangiectasis) and Associated Cerebral Movements." Discussed by Drs. H. H. Hoppe, Cincinnati, and William G. Spiller, Philadelphia.

Dr. Leo M. Crafts, Minneapolis, read a paper on "Original Test for the Pathologic Great Toe Sign with Illustrative Cases." Discussed by Dr. George A. Moleen, Denver.

Dr. Tom B. Throckmorton, Des Moines, read a paper on "Clinical Report of a Case of Tumor of the Pons Varolii." Discussed by Drs. H. H. Hoppe and F. W. Langdon, Cincinnati.

Drs. Peter Bassoe and George B. Hassin, Chicago, presented a paper on "The Histologopathology of Epidemic ("Lethargic") Encephalitis." Discussed by Drs. Max H. Weinburg, Pittsburgh, and Peter Bassoe, Chicago.

Dr. Beverly R. Tucker, Richmond, Va., read a paper on "The Rôle of the Pituitary Gland in Epilepsy." Discussed by Drs. Walter Timme, New York; H. H. Hoppe, Cincinnati; E. B. Angell, Rochester, N. Y.; George A. Moleen, Denver; Albert E. Sterne, Indianapolis; Irving J. Sands, Brooklyn, and Beverly R. Tucker, Richmond, Va.

FRIDAY, JUNE 13—AFTERNOON

In the absence of the chairman, the meeting was called to order at 2 o'clock by Dr. H. H. Hoppe, Cincinnati.

Dr. Harry H. Drysdale, Cleveland, read a paper on "A Case of Hysterical Hemiplegia Following a Shrapnel Wound of the Scalp, and Presenting Interesting Clinical Features." Discussed by Drs. Alfred Gordon, Philadelphia; Tom A. Williams, Washington, D. C.; Albert E. Sterne, Indianapolis;

Digitized by Google

Lewis J. Pollock, Chicago; Hugh T. Patrick, Chicago; W. S. Lindsay, Topeka, Kan.; Foster Kennedy, New York; Edward E. Mayer, Pittsburgh; Julius Grinker, Chicago, and Harry H. Drysdale, Cleveland.

Dr. Alfred Gordon, Philadelphia, read a paper on "Multiple Neuritis of Toxi-Infectious Origin with Especial Reference to Diabetic Polyneuritis." No discussion.

Dr. George Wilson, Philadelphia, read a paper on "The Resemblance of the Sensory Symptoms of Post-Diphtheritic Ataxia to Those Seen in the Cord Changes of Severe Anemia." Discussed by Dr. W. W. Richardson, Mercer, Pa.

Drs. William H. Robey, Jr., and Heiman Caro, Boston, read a paper on "Neurologic Sequelae Following Acute Respiratory Infections." Discussed by Drs. Charles A. Rosewater, Newark, N. J.; W. H. Mayer, Pittsburgh; Max H. Weinburg, Pittsburgh; Alfred Gordon, Philadelphia, and William H. Robey, Jr., Boston.

Digitized by Google

Digitized by Google

THE NEUROLOGY OF CERVICAL RIBS *

ARCHIBALD CHURCH, M.D.
Professor of Nervous and Mental Diseases, Northwestern University Medical School
CHICAGO

Supernumerary cervical ribs, as furnishing the cause of many obscure and some very serious nerve lesions affecting the arm, have not been sufficiently recognized in neurologic literature. It is almost startling that no textbook on nervous diseases makes the slightest detailed mention of cervical ribs in connection with brachial plexus disorders, excepting the English translation of Oppenheim. Even the great handbook of Levandowsky has only a slight reference to cervical ribs, and Chipault's "Surgery of the Nervous System" is devoid of any mention of the subject. Works on surgery and radiography have much more.

The frequency of cervical ribs is much greater than is usually understood. Fischel in Prague, in four years, found cervical ribs in 1 per cent. of all the bodies examined. Their most common location is in conjunction with the seventh cervical vertebra, next in relation to the sixth and seventh, and then to the fifth and fourth in descending order of frequency. They have never been observed attached to the first, second or third.

Historically, the cervical rib has been well represented for more than 150 years. Its first description was by Hunalt in 1740, and it was again fully described by Gruber in 1869. In 1894, Pilling collected seventy-six instances. Its first recognition during life was apparently by Cooper in 1818, and it was first surgically extirpated in 1861 by Coote. Since we have had the use of the roentgen ray it has often been unexpectedly found, and not infrequently the roentgen ray adds

* Chairman's address.

Digitized by Google

proof positive to the clinical diagnosis of cervical ribs, which in a general way can be rather definitely diagnosed from the nervous and vascular symptoms they sometimes produce. The very best paper on the subject of cervical ribs is that of Streissler,[1] who considers the subject from every point of view, giving 297 references and 200 cases.

Somewhat strangely in view of the fact that the female organism is supposed to be more stable and less subject to variation than that of the male, 63 per cent. of the cases of cervical ribs are found in women according to Walther, while Streissler found 70 per cent. female and 30 per cent. male.

Of course cervical ribs are congenital; but clinical manifestations rarely develop under the age of 10, commonly make their appearance between 10 and 30, and occasionally give rise to symptoms which first appear only in the later decades of life.

The supernumerary ribs are bilateral in a little more than half the cases, and when lacking in symmetry or represented only on one side, have a disposition to appear more frequently on the left. Occasionally they show hereditary tendencies or constitute a family marking. Thus, Russell[2] found them in sisters, Drehmann[3] in a family presenting eighteen cases, and I have seen them in a mother and daughter.

The disturbance occasioned by cervical ribs does not bear any definite relation to the size of the rib, some of the largest occasioning no symptoms at all, and some comparatively small ones being attended by very disabling and even destructive processes affecting the upper extremity.

Gruber is disposed to classify cervical ribs, according to their size, into four groups:

---

1. Streissler, E.: Die Halsrippen, Ergebn. d. Chir. u. Orth. **5**: 280, 1913.
2. Russell, C. K.: M. Rec. **71**: 253.
3. Drehmann, G.: Cervicodorsal Scoliosis and Supernumerary Ribs with and without Supernumerary Cervical Rudimentary Remnants, Ztschr. f. orthop. Chir. **16**: 12; Heredity and Family Groups, Ten Cases, Allg. med. Centr. Ztg., 1905, No. 2, p. 21.

Digitized by Google

1. Consisting only of a node which does not extend beyond the lateral dimensions of the transverse processes of the vertebra.
2. A blunt finger of bone 4 or 5 cm. in length.
3. A rib which extends far enough forward to articulate with the first rib or even to be attached to the sternum by a ligamentous cord.
4. A complete rib having vertebral origin and costosternal cartilage.

These ribs are commonly associated with other skeletal abnormalities, especially those marking the sternum, the vertebrae and the ribs. The articulation of the cervical rib to the vertebra either is in the form of a full synostosis, or in some cases the union is partially osteal. Modifications of the scalene muscles are occasioned by the projection of this adventitious bony development, but the subclavian artery always curves above the rib or the fibrous cord, which extends from the tip of the rib forward to the sternum and which in certain cases causes as much difficulty and as much damage as a thoroughly developed bone.

The thorax is changed in its conformation to some extent, having higher apexes with salient arteries readily palpable above the level of the clavicle; sometimes visible pulsation can be made out in the triangles of the neck. This unusual extent of the pulmonic tissues and the feeling of bony hardness or osteal intumescence is a frequent source of mistaken diagnosis.

ETIOLOGY

The disturbances arising from the presence of cervical ribs, which commonly appear only after the end of the first decade of life, seem to be brought about by the naturally increasing rigidity of the bony and cartilaginous structures, reduction of elasticity of arteries and nerves, and the increased weight thrown on the upper extremities in the various activities of life. Occasionally the disorder in the arm is induced by traumatism, such as bruises or pressure occurring from carrying heavy weights on the shoulder or in the hands. Frank[4]

4. Frank, O.: Symptoms Induced by Carrying Milk Yoke, Direct Violence, Deutsch. med. Wchnschr., 1907, No. 14, p. 597.

Digitized by Google

reports a case in which a yoke used for carrying milk pails brought about disturbance in the arms in the case of a milkmaid who had cervical ribs, and Streissler has seen a case in which carrying a gun on the shoulder led to the development of disorder in the hand. In one of my cases, symptoms followed long continued efforts at cranking a gas engine, and in another, unusual lifting of heavy objects preceded the onset of symptoms.

Tuberculosis affecting the pulmonary apex has of course been found in frequent association with cervical ribs, and in some instances the contiguous inflammation seems to have disturbed the brachial nerve trunks in their passage over this adventitious structure. So pleurisy, exudation, and occasionally the loss of fat, thereby laying the parts subject to more intimate injury, have led to the development of these peculiar disorders. The fracture of the clavicle and the development of callus, encroaching on the space between the shoulder girdle and the thorax, have been the initial features in some cases. Periostitis of the supernumerary rib has occurred, and occasionally it is the seat of tumor growth. Nerves which are exposed to the disadvantage of passing over an additional rib, under general systemic toxic conditions, such as those associated with diphtheria and arthritis, have shown an additional vulnerability; and disorder in their peripheral distribution has subsequently followed. So, too, arteriosclerosis of the subclavian, which is in close apposition to the brachial roots above the supernumerary rib, can seriously affect these nerve structures as well as cause vascular faults in the hand.

SYMPTOMATOLOGY

As already indicated, the symptomatology of disorder of the nerves and vessels due to cervical rib is not relative to the size of the accessory structure. These symptoms consist first of local signs. In some instances a tumor-like mass can be made out at the side of the neck, behind the clavicle, at the apex of the thorax;

Digitized by Google

but the outline of the supernumerary rib cannot probably be ever determined by palpation. A more than usually salient subclavian artery and general widening of the root of the neck and a lofty lung apex are objective in character and capable of detection by the usual means of observation. The roentgen ray furnishes a very definite method of detecting these cervical ribs, but a certain amount of experience is essential in interpreting the plates. Cervical ribs of the first order laid down by Gruber are so clearly parallel with the transverse process that unless the negative is particularly definite and scrutinized with great care, a cervical rib of dimensions which do not carry it beyond the tip of the transverse process may be entirely overlooked. I know of a prominent surgeon who from the roentgenogram determined the presence of a cervical rib which on operation was not found; and subsequent roentgenographic examinations failed to show anything like the artefarct which was present in the original negative and which was certainly misleading. Again, when the supernumerary ribs are well developed and tolerably symmetrical, they may be mistaken easily for the first thoracic pair, and it is only by counting the cervical vertebrae and by lateral views that their exact nature can be determined. A rudimentary first dorsal rib has been mistaken for a cervical rib.

VASCULAR CHANGES

While vascular changes are not so common as nerve disturbances, as a rule, in some cases they are very prominent. A change may be found in the character of the pulse as compared with the sound side, and certain positions given the upper extremity may serve to stop the pulse altogether, as, for instance, in the case of a soldier whose pulse failed always when he carried his gun on any part of his shoulder, and equally could be extinguished by raising his hand to the same position. Sometimes by placing the hand above the head

Digitized by Google

or by drawing down on the extremity, as in carrying burdens in the hands, the pulse can be reduced in amplitude and even extinguished. In other cases the pulse will vary with respiration, tending to disappear on full inspiration. In rare cases, disturbances of the subclavian by the pressure of the rib has resulted in thrombosis and loss of all pulse in the extremity. In other cases, aneurysm has formed at the point where the subclavian passes over the rib. Several of these conditions are competent to produce an inequality of the radial beat in the two extremities.

The peripheral circulation in the extremity is very frequently disturbed, and some cases of miscalled Raynaud's disease have been due to cervical rib. The fingers often present a blue, edematous or reddened condition, and this may be accentuated by the involvement of the nerves and the trophic changes and glossy skin that attend a neuritis. Many patients complain that the hand on the affected side is much more influenced by cold, and that the hand is invariably colder than the opposite one. Intense edema, vasomotor flushing, and hyperhidrosis are common in the severe cases.

NERVOUS SYMPTOMS

Nervous symptoms are the most common features in cases of cervical rib that affect the upper extremity. The sensory defect may be confined to the distribution of a single nerve or may embrace the entire member; and pains of a neuralgic character may radiate to the back of the neck, the back of the shoulder, the neighborhood of the ear, very frequently down the side of the chest in the axillary line, and in some cases are complained of over the entire side of the body. The sensory disturbance is usually most intense in the fingers and most commonly in the ulnar portion of the hand and in the ulnar fingers, although in a fair proportion of the cases the radial distribution in the thumb and index is the seat of the greatest complaint. Pares-

Digitized by Google

thesias, hypersensitiveness and hyposensitiveness of all grades may be found in varying cases, and Barker[5] has from personal experience called very definite attention to dissociation of sensation in the affected member similar to that found in syringomyelia. On the motor side weakness is very common, and if the painful manifestations are pronounced, disuse of the arm leads to a still greater weakness. Very rarely is there complete paralysis. All varieties of claw hand may be encountered, with the corresponding contractures due to the involvement of the roots of the brachial plexus in varying degree or in its entirety. There are commonly functional difficulties in the use of the hand, which sometimes in a minor measure are present only in the finer manipulations, such as writing. These patients usually experience inconvenience and sometimes intense pain when carrying burdens in the hand or on the arm, and frequently adopt a sling that supports the weight of the member and relieves the drag on the injured nerves and vessels at the point of pressure.

TROPHIC DISTURBANCES

Disturbances of the trophic control in the small muscles of the hand and the forearm are very common, and usually the muscle group originating from the internal condyle is the one most involved in the forearm. With this may be found trophic disturbance of the skin, especially at the finger ends, trophic blebs, glossy skin, changes in the nails, and hemorrhages under the nails. Occasionally the sympathetic nerve has been involved, giving rise to pupillary disturbance, ptosis, and retraction or protrusion of the eye. The phrenic nerve may have been occasionally affected.

In quite a number of cases, hoarseness is reported. Whether or not the right recurrent nerve which loops under the subclavian at the point where the subclavian arches over the supernumerary rib is ever involved is

5. Barker, L. F.: A Case of Unilateral Circumscribed and Elective Sensory Paralysis, J. Exper. M. 1896; Deutsch. Ztschr. f. Nervenh. 8: 348, 1896.

Digitized by Google

a somewhat disputed point. Streissler thinks it doubtful. Planet[6] described a case in which there was hoarseness, and Dr. Carl Davis of Chicago has reported two such cases. Weisenstein[7] reported a case in which a patient was aphonic every morning. This seems to have been hysterical rather than due to involvement of the recurrent. A similar case is reported by Ranzi.[8] Spiller and Gittings[9] noted one case in which there was atrophy of the tongue and larynx. I myself have had under observation an otherwise healthy woman who has a symmetrical pair of cervical ribs, and who presents at times paresthesia and numbness in the hands, particularly the right. She has had three very prolonged attacks of aphonia attended by paresis of the right vocal cord. At least two of these attacks have been brought on by overexertion in the way of lifting, and I think, after a faithful search for other causes, are undoubtedly due to involvement of the recurrent by the cervical rib.

CERVICAL SCOLIOSIS AND CERVICAL RIBS

Ever since Garrè[10] called definite attention to the presence of cervical ribs, usually asymmetrical or unilateral, in conjunction with cervical scoliosis, the frequency of these observations has been increasing. Streissler found such a scoliosis in 16 per cent. of his cases. Frequently the cervical deviation is attended by more or less asymmetry of the face and of the skull, and of course is attended by compensatory curves in the dorsal region. The deviation of the spine does not always seem to be proportionate to the size of the supernumerary rib; but sometimes a supernumerary wedge-shaped cervical vertebra is found to which the

6. Planet: Tumeurs osseuses du cou, la septième côte cervicale, Thèse de Paris, 1890.

7. Weisenstein: Zur klinischen Bedeutung der Halsrippe, Wien. klin. Rundschau, 1903.

8. Ranzi, E.: Zur Kasuistik der Halsrippen, Wien. klin. Wchnschr., 1902.

9. Spiller and Gittings: Muscular Atrophy of Cervicobulbar Type and Cervical Rib; Tongue and Larynx Muscle Atrophy; Shoulder Girdle and Upper Extremities Affected, New York M. J., Oct. 6, 1906.

10. Garrè: Four Cases of Cervicoscoliosis and Cervical Ribs, Ztschr. f. orthop. Chir. 9: 73.

Digitized by Google

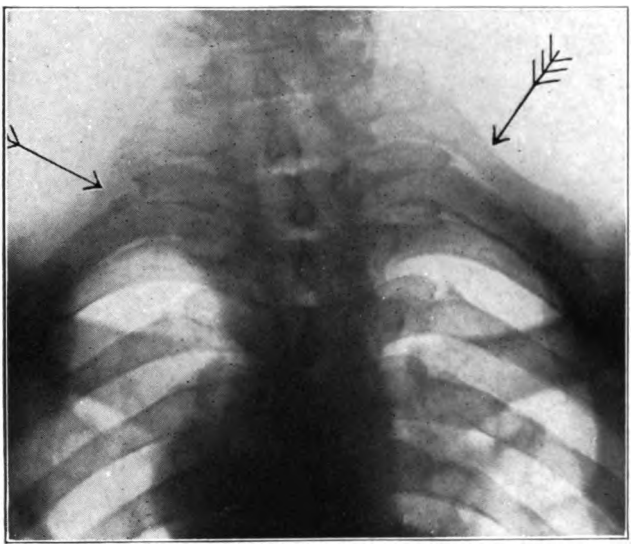

Fig. 1.—Very large right cervical rib, smaller left cervical rib, indicated by arrows. Cervical scoliosis with compensatory curves. Not attended by any vascular or nervous symptoms.

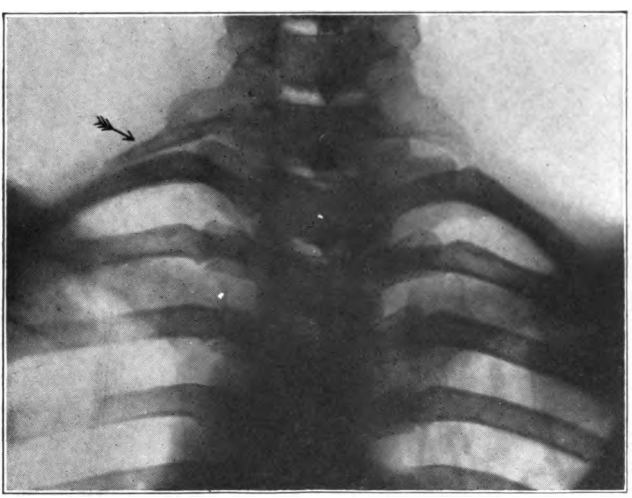

Fig. 2.—Bilateral cervical ribs, right one of the first degree, left one of the second degree, according to Grubbe. (Courtesy of Dr. Hollis E. Potter.)

Digitized by Google

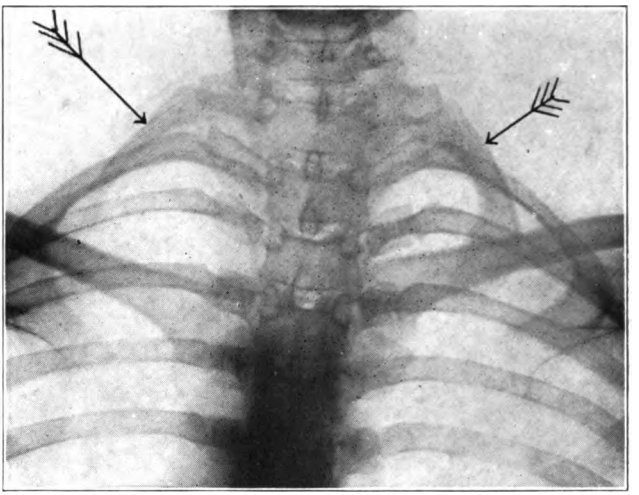

Fig. 3.—Large asymmetrical cervical ribs, indicated by arrows. Neuritis and vascular disturbances in the right arm. Very slight cervical scoliosis.

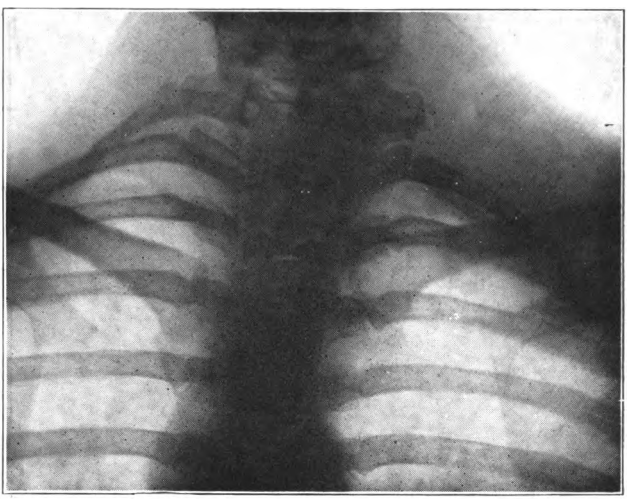

Fig. 4.—Same patient as in Figure 3 after operation for the removal of the right supernumerary cervical rib.

Digitized by Google

supernumerary rib is attached. Of course such a scoliosis is attended by some postural displacement of the shoulder and scapula. In every case of cervical scoliosis, supernumerary ribs should be suspected.

DIAGNOSIS

The diagnosis of cervical ribs sometimes is attended with a great deal of difficulty, and a large number of other conditions can easily be confused with them. Tumors in the angles of the neck or growing from the cervical and upper dorsal vertebrae may bring about similar conditions in the related extremity. Aneurysms of the subclavian produce a similar symptom group. Quite a number of cases of Raynaud's disease have been found not to be due to the vascular disorder of symmetrical gangrene, etc., but to have been caused by the vascular and neuritic changes secondary to cervical ribs. As the cervical rib produces a brachial neuritis, its presence has been overlooked in this rather common disorder, the neuritis being attributed to systemic or local conditions of a different order; and so neuralgias affecting the upper extremity have perhaps in many instances been of mistaken origin. Subacromial bursitis and arthritis of the shoulder joint producing painful disability of the upper extremity might easily be confused with the effects of a cervical rib. Several cases have been diagnosed as pachymeningitis cervicalis, others as poliomyelitis, others as progressive atrophy, and still others as syringomyelia. Further, owing to the involvement of the thoracic branches of the cervical plexus, some of these cases have been confounded with intercostal neuralgias, and the cervical scoliosis has for some furnished an acceptable explanation of the changes in the hand, without recognition of its association with and dependence to a large degree on the presence of cervical ribs. Dulness and conformation peculiarities of the lung apex have led to a diagnosis of phthisis, and of course their association,

Digitized by Google

in view of the prevalence of phthisis, is not unusual. If one, however, has in mind the frequency of cervical ribs and their capacity for producing painful vascular and trophic changes and symptoms in the upper extremity, careful search of the case with the aid of the roentgen ray will usually make the diagnosis quite definite.

COURSE AND PROGNOSIS

Many cases show long periods of symptoms or even of very decided disability, with intervals of camparative or complete freedom. Nevertheless, the course of the disorder in the arm is usually protracted and sometimes continues for many years. Nerves and vessels placed at the disadvantage caused by the peculiar anatomic variation presented by cervical ribs can be easily injured in ordinary life pursuits and may recover under conditions of rest and protection. The case histories of numerous severe instances indicate that the tendency is for the disease to come on insidiously, to advance steadily, and to reach such a degree that amputation of the hand or arm or other heroic surgical intervention is readily accepted. The very severe cases are practically hopelessly short of surgery, which has for its proper object the removal of the cervical rib.

TREATMENT

In the milder cases a conservative treatment is altogether advisable. Rest, avoidance of strain and weight carrying, and the thorough support of the arm by a properly arranged sling all tend to avoid irritation of the nerves and vessels by rib contact. Electricity, massage, applications of heat and cold, and general measures, such as are commonly indicated in a brachial neuritis, have their very definite place in the management of these cases, but in the severer ones give rise to so much pain and inconvenience as to be almost insupportable. In cases of the more pronounced grade, recourse to surgery is the only avenue to cure.

Digitized by Google

The extirpation of a cervical rib constitutes one of the most difficult of major surgical measures, and must not be lightly undertaken. Not only does it require a very difinite anatomic dissection, but dissection in a very dangerous location that is rendered more dangerous by unusual anatomic relations. A number of surgical mishaps have occurred. Jones[11] of Manchester reports injuries to the arteries, to the pleura, and to the subclavian vein. In a number of instances the incomplete removal of the cervical rib has been followed by its regrowth from the proximal stump and the reappearance of all the former disabilities. It seems to be highly important that the periosteum should be completely removed. In other instances, sufficient attention has not been given to the cordlike extension which represents the anterior portion of the incomplete rib, and symptoms have consequently not been relieved. When complete extirpation can be effected, the prospect for full relief and restitution of function is practically perfectly good; but chronic neuritic, vascular and trophic processes, often attended by contractures, require months and sometimes years of time and treatment to recede entirely.

My own experience is based on eleven cases, all of which have been verified either by operation or roentgenoscopy or both, and all of which have been relieved with one exception. In this instance a very competent surgeon removed most of the cervical rib. I must reiterate the view of Miller,[12] who insists that in every case of protracted discomfort, pain, or trophic disturbance in the arm, a careful search should be made for cervical ribs. The same advice applies to every case of cervical scoliosis.

206 South Michigan Avenue.

11. Jones, H. L.: Difficulty of Extirpation and Harm to Plexus; Recurrences by New Bone Formation from Stump, Quart. J. Med. **1**: 187.

12. Miller, J. L.: Observations on the Symptomatology and Diagnosis of Cervical Rib, Am. J. M. Sc. **142**: 811 (Dec.) 1911.

Digitized by Google

ADDITIONAL REFERENCES

Babcock, W. W.: Cervical Rib with Resulting Gangrene of the Fingers, Surg., Gynec. & Obst. **2**: 113, 1906.

Beck, C.: Surgical Importance of the Cervical Rib, J. A. M. A. **44**: 1913 (June 17) 1905.

Bernhardt, M.: With Basedow's Disease, Berl. klin. Wchnschr., No. 27, p. 905; Postoperative Palsies, Serratus, Deltoid and General Brachial Distribution, Berl. klin. Wchnschr., No. 31, p. 1441.

Borchardt, M.: Symptomatologie und Therapie del Halsrippen, Berl. klin. Wchnschr. **38**: 1265-1271, 1901.

Flatau and Sawicki: General Description, Mention of Difficult Deglutition, Arb. des. I. Kong. poln. Neurologen u. Psychiater.

Francine, A. M.: Foru Cases, Two Flail-like, Am. J. M. Sc. **139**: 1-8.

Goodhart, S. P.: Cervical Rib and Its Relation to the Neuropathies, Am. J. M. Sc. **138**: 666-683, 19009.

Gordon, . E.: Brit. M. J. **1**: 1395, 1900.

Helbing, C.: Scoliosis with Cervical Ribs, Ztschr. f. orthop. Chir. **12**: 216.

Hessert, William: Cervical Rib, Ann. Surg. **44**: 630, 1906.

Kammerer, F.: Cervical Ribs, Ann. Surg. **34**: 637-648, 1901.

Keen, W. W.: The Symptomatology, Diagnosis and Surgical Treatment of Cervical Ribs, Am. J. M. Sc. **133**: 173-218, 1907.

Levi, H.: With Multiple Sclerosis and Syringomyelia, Neurol. Centralbl., No. 21, p. 988, 1903.

Marburg: With Syringomyelie, Wien. klin. Rundschau, No. 13, p. 241, 1905.

Mendel, K.: Neurol. Centralbl., No. 9, p. 556, 1914.

Meyerowitz, F.: Scoliosis from Cervical Rib, Inaug. Diss., Königsberg, 1904.

Murphy, J. B.: Clinical Significance of Cervical Ribs, Surg., Gynec. & Obst., October, 1906; Case of Cervical Rib with Symptoms Resembling Subclavian Aneurysm, Ann. Surg. **41**: 398-406, 1905.

Nicol, W.: Association with Apical Lung Infiltration, Dissertation, Leipzig.

Osler, William: Certain Vasomotor, Sensory and Muscular Phenomena Associated with Cervical Rib, Am. J. M. Sc. **139**: 469-472, 1910.

Riesman, D.: Cervical Rib, Univ. Penn. Med. Bull. **17**: 2, 1904-1905.

Schtscherbak and Kaplan: Obszr. psichiat., nevrol., No. 21, 1907.

Seymour, N. G.: Am. J. M. Sc. **146**: 396, 1913.

Thomas, G. F.: Undeveloped First Dorsal Rib Simulating a Cervical Rib. J. A. M. A. **52**: 1405 (May 1) 1909.

Weber, F. P.: Family History of Cervical Ribs, Tr. Med. Soc., London, **32**:

Weiss, Hugo: Die Halsrippen und ihre klinischen Erscheinungen, Centralbl. f. d. Grenzgeb. d. Med. u. Chir. **3**: 897-900, 1900.

Williams, H. J.: Report of Case, with Remarks on Mistaken Skiagraphical Diagnosis, Ann. Surg. **28**: 509-515, 1898.

Digitized by Google

TRAUMATIC NEUROSES IN WAR AND IN PEACE

T. H. WEISENBURG, M.D.
PHILADELPHIA

The chief story of the war neuroses has already been written in an excellent paper by Major S. I. Schwab, which appeared in the April number of the *Archives of Neurology and Psychiatry*. Dr. Schwab was chief medical director of Base Hospital No. 117 in France throughout its entire history. It is apparent from his paper, and those of other neurologists who had experience with war neuroses, that while a great deal that was new was observed, yet no startlingly new facts were developed. The work of Dr. Schwab and others was chiefly concerned with the study of the neuroses, either immediately after combat or within a few weeks after their origin. It would not, therefore, be uninteresting to record a study of the war neuroses as they appeared in this country.

For a period of four months I was on duty in U. S. Army General Hospital No. 30, Plattsburg Barracks, N. Y. This hospital was the only so-called war neuroses hospital conducted by the War Department in this country. There were three groups of patients: (1) those who had been sent from camps in this country; (2) soldiers who had been in France but who had not seen any actual fighting, and (3) soldiers who had been in combat. The majority of these cases, no matter what their source, were sent to Plattsburg because they were difficult to cure.

This was particularly true of the overseas cases. It is, of course, known that a majority of the patients

Digitized by Google

were cured in front line stations, and that those who were not thus cured were sent to Base Hospital No. 117 or to other centers. The more refractory of these cases or those that seemed to be incurable, or that for some reason or other did not offer favorable conditions for cure, were returned to the United States and to Plattsburg.

In this paper, no attempt will be made to describe at length the different types of neuroses met with, or to enter into a discussion of the mechanism producing them, its purpose being merely to compare the type of neuroses met with under war conditions and in civil life.

HYSTERIA

Of the patients studied in Plattsburg, those with hysteria attracted most attention. There were present the usual types which have been so well and often described, such as aphonia, stuttering, amnesias, paralyses of both legs and more rarely of one upper or lower limb, less frequently a combination of mutism with deafness, a few cases of blindness, tremors, gaits of various sorts, and a large number of so-called epileptic or fainting cases. There is no need of describing the sypmtoms in these cases, for the patients presented the usual pictures which are present in all. However, some unusual phenomena were observed. There was one case, which is worth particular mention, of a man who had a constant dancing of the eyeballs:

A boy, aged 18 years, with a normal history, after a particularly trying attack which lasted many days, and in which many of his companions were killed, began to develop a tremor all over his body with quick jerking movements of the head and a constant dancing of the eyeballs to which his lieutenant called his attention. This was in April, 1918. His tremors gradually lessened, but the movements of the eyeballs continued. It is interesting to note that besides having been gassed he had a flesh wound in the left leg, and a rifle bullet wound in one shoulder. Examination did not detect anything unusual, with the exception of quick reflexes; but there was present a constant horizontal nystagmus, which greatly

Digitized by Google

increased under excitement, and almost disappeared when the patient was at rest or when free from exciting stimuli, and was absent when he was asleep. The ocular movements were quick and of wide range, and were different from the nystagmus seen in multiple sclerosis or any other disease.

There is no reason why there should not be a hysterical tremor in the eyeballs, just as well as in any other part of the body, but that such a thing is possible, is interesting.

EPILEPSY AND HYSTERIA

One of the most difficult problems was the differential diagnosis of epilepsy from hysteria. It was commonly known by the soldier that fits would disqualify him for service and as a consequence, a great many so-called epilepsies developed. Of those patients returned to Plattsburg from the American Expeditionary Forces whose cases were diagnosed as epilepsy, a surprising number proved to be such, although there were a great many cases of hysteria diagnosed as epilepsy. On the whole, it cannot be said that war experience produced epileptic attacks; but undoubtedly in a number of patients, epilepsy appeared earlier than would have been the case in ordinary civil life. The diagnosis of epilepsy was nearly always possible after an attack was observed, although in a number of cases it was difficult.

There was no essential difference in the hysterias which developed in the patients who had had their entire service in this country, and in those who had been with the American Expeditionary Forces, including those who had been in actual combat, with the exception that in the home troops tremors were not prominent. There were several cases of so-called "trench back" or camptocormia, all of them from this country. There were three instances of so-called reflex contractures in the hands and fingers. As is, of course, well known, Babinski and Froment believe that these are organic in character; the opposing view being held

Digitized by Google

by others, notably Roussy and Hurst. In the few cases observed by us, we were able to effect a cure, and I have no doubt that they were purely hysterical in character.

It has been said that this war produced two new types of neuroses: (1) the so-called reflex contractures of the hands and fingers, and (2) trench back or camptocormia. I have seen some examples of the latter condition in civil life, and in January, 1918, I exhibited in a clinic, to a class of medical officers under instruction at that time in Philadelphia, a medicolegal case with a history of an injury to the hand, a typical example of reflex contracture of the hands and fingers.

SIMILARITY OF NEUROSES IN WAR AND IN PEACE

In view of this experience, the question naturally arises: Is there any difference between the hysterias of war and of peace? In my experience of a good many years with traumatic neuroses I have seen a duplication of practically every type of hysteria seen in Plattsburg, but, of course, not in such abundance and of such variety. In civil life, aphonia, stuttering, deafness and blindness are not so frequent; but paralysis, contractures, tremors and fits are common.

This view is not held by Schwab, who states that "the war neuroses are specific war conditions, and with the ending of the war they disappear from clinical experience. Their place is taken by the civilian neuroses, which are different things." The opposing view is taken by Leri, who believes that the hysterias of war and of peace are identical.

This difference in the point of view held by Schwab and by myself is, no doubt, due to the different conditions under which neuroses were studied. Many of the patients returned from France were so much better when they reached Plattsburg that they presented an entirely different clinical picture. Besides, their presence in this country virtually assured them that they

Digitized by Google

would not be returned to France. Therefore the conditions surrounding them approximated a peacelike basis and offered an excellent opportunity for comparison.

Of the neuroses, other than hysteria, Schwab mentions neurasthenia; psychasthenia; hypochondriasis; anxiety neurosis; effort syndrome; exhaustion; timorousness or state of anxiety; concussion, (*a*) syndrome, (*b*) neurosis; gas, (*a*) syndrome, (*b*) neurosis.

As in hysteria, the clinical picture of these conditions changed materially, and only the neurasthenias and anxiety neuroses were at all prominent, although there were some psychasthenic and hypochondriac states. These neuroses were similar to those found in civil life.

INFLUENCE OF THE ARMISTICE

Some interesting observations were made regarding the suggestibility of patients and the influence of the armistice. It did not cause a very great change, and there were practically no spontaneous cures, although our work became much easier and there was a greater incentive for the patients to get well and less difficulty in getting them well. One interesting observation was made: Before the armistice it was possible to suggest new areas of disturbance of sensation or an increase of paralysis. This was impossible after the armistice, and the only suggestion that the patients took kindly to was that of getting well.

The suggestibility of hysteria was very well shown in the occurrence of "fits." Patients in whom attacks were absent for months suddenly developed them in times of stress; as, for example, when under arrest after an offense had been committed. One patient left the post under the impression that he had the right to do so, but on discovering that he was absent without leave, he developed a series of attacks lasting several days, giving this occurrence as an excuse for his absence.

Digitized by Google

PERSONALITY OF THE WAR NEUROTIC

There was, however, one very distinct impression made on all of the medical officers who were on duty at Plattsburg for any length of time. It was the first time that I have ever had the opportunity of living exclusively among neurotics, for any length of time. Because of the peculiar situation at Plattsburg, it being an old army post converted into a hospital with practically no outside attractions, the atmosphere of the place naturally was much more acutely felt than would have been the case if such a hospital had been located in or near a large city. I was sent to Plattsburg and was placed in charge of all treatment; that is, it was my duty to see that the patients got well. The patients were indifferent. They seemed to care about getting well and yet not to a very great extent. After the armistice, it was different. They wanted to go home, but the incentive for getting well was not even then what it should have been. The individual patients, while they were talkative, especially about their deeds of valor, at the same time were by no means so cheerful, openhearted and joyful as the average wounded soldier in other hospitals. Most of the patients were boastful, and wore all sorts of decorations, even wound stripes to which they were not entitled. On questioning, it was often found that some had put on wound stripes after they had been in a hospital with a mere cold.

Another noticeable thing was the difficulty we encountered in managing both enlisted men and officers. They were constantly complaining and finding fault. There were many who had been absent without leave. Every one who is familiar with the service knows that all soldiers grumble about everything under the sun. This is a normal condition; but the patients in Plattsburg were certainly more difficult to handle than were those in other hospitals. Perhaps this should be expected among such unstable neurotics.

Digitized by Google

In other words, the conclusion was forced on me that the war neurotic, as seen in Plattsburg, was distinctly different from the normal individual; that he was boastful, somewhat depressed, erratic and certainly unreliable.

Numerous studies have been made of the personality of the war neurotic and of his neuropathic inheritance. Schwab states that the proportion of neuropathic cases in his experience was never under 2 per cent., or much greater than 5 per cent. When, however, it is taken into consideration that the more difficult and intractable cases were returned to the United States, it can be readily seen that the proportion of such individuals was much greater in Plattsburg.

TREATMENT OF THE WAR NEUROTIC

The treatment of the war neurotics in Plattsburg did not offer any unusual experience. The method adopted was similar to that found successful in neurologic centers abroad. The physicians, hospital corps men and nurses were instructed as to the nature of hysteria and war neuroses, and an effort was made to institute a so-called "atmosphere of cure," which was eminently successful. Electricity was only occasionally used. In fact, cases which were difficult to cure in France and in camps in this country offered no special difficulty, once the "atmosphere of cure" was established; for as happens in such instances, success led to success, and nurses and hospital corps men were as successful as the physicians.

As an example of this, I lectured one afternoon to those hospital corps men who were on duty at night. That same evening, twelve patients arrived from Camp Taylor, and were met by one of these men. Among these patients was a man who had paralysis of an arm, another who could not talk, a third who walked in a hopping manner, a fourth with paralysis of a leg, and a fifth with a very markedly bent back. On the way

Digitized by Google

to the hospital, a hospital corps man told the patients of the wonderful cures that were made. I saw these patients on their arrival at the hospital and, seeing the good work that was being done by the hospital corps men, gave them instructions not to have them disturbed. The following day all these patients were well with the exception of the man with the bent back, who after one application of the faradic current promptly straightened up. This is an example of what can be done by an untrained attendant where there is good team work and the example of success.

TREATMENT OF THE PEACE NEUROTIC

The treatment of the traumatic neuroses presents different conditions. In the war neuroses, as has been generally accepted, the cause lies in a defense reaction to a personally intolerable situation, whereas in the peace traumatic neuroses, the causes can be assigned either to the desire of the injured for compensation, in the actual trauma produced, or in the fear of the consequences of the injury. I have had many years of experience with traumatic neuroses, testifying both for and against corporations, and I have always felt that the chief reason why so many of these cases last such a long time is that they are not disposed of immediately after their occurrence.

In the work of the Industrial Accident Commission of California, an excellent opportunity has been offered to study the final outcome of accident neuroses. Under the direction of Dr. Walter F. Schaller,[1] a special investigation was made of fifty cases. All but four of the persons were personally interviewed, and in all but two of them the factor for recompense was present. It is unnecessary to give in detail the results of this investigation, but it is interesting to note some salient facts:

1. Schaller, W. F.: Diagnosis in Traumatic Neurosis, J. A. M. A. 71: 338 (Aug. 3) 1918.

Digitized by Google

Age was apparently unimportant.

Women showed a greater tendency to recover, although not one was entirely relieved.

The severity of the original injury apparently bore no relation to the outlook for recovery.

Most of the symptoms were predominantly hysterical.

The cheerful patients showed a marked contrast in recovering to those who were depressed.

Schaller emphasizes that of all the factors which enter into the course of the accident neuroses, the question of compensation is one of the most important, and its great psychic influence cannot be denied. This was not true, however, in all of his cases, for in three, there was recovery while the patients were still being paid under the periodic payment plan. The cases showing lump sum settlement proved the most favorable for recovery, early lump sum settlement being more favorable than periodic payments and a final lump sum settlement.

One very interesting phase of the investigation was the lapse of time between the injury and the recovery. the longest period being three years and nine months and the shortest, seven months. It can be seen from the foregoing that there is no basis of comparison, so far as cure is concerned, between the traumatic neuroses of war and of peace.

1909 Chestnut Street.

Digitized by Google

THE MANAGEMENT OF WAR HYSTERIA

TOM A. WILLIAMS, M.D.
WASHINGTON, D. C.

Functional nervous disease is responsible for at least 10 per cent. of the evacuations of soldiers during an attack. The vast majority of these men are found to have hysteria, and the greater proportion of them, if properly managed at once, can be returned to the firing line in a state of greater efficiency than before their sickness.

Those who do not have hysteria are for the most part in a state of high emotivity, termed "anxiety neurosis" on the records of the American Expeditionary Forces. However, the majority of anxiety cases do not arise during battle. That condition is usually of slow onset, and occurs after long-continued duty, as has been shown in the French army.

EXCLUSION OF COMMOTION

A small proportion of the men evacuated during battle will be found to have cerebral commotion, true shell shock. This is characterized by: (1) the history of long continued coma followed by torpidity and inertia; (2) a period of obnubilation and confusion often accompanied by hallucinations, delusions and erratic behavior; (3) modification of the reflexes; the cutaneous reflexes may be absent and the plantar reflex in extension; the tendon reflexes may be exaggerated, diminished or asymmetrical at different periods; the pupils are usually widely dilated, although they react except during the initial coma; (4) vasomotor disturbances; palor, cyanosis, hypotension with asym-

Digitized by Google

metry in different limbs; (5) alterations in the cerebrospinal fluid, characterized by a disproportionate abundance of serum-globulin and albumen, with a relatively small increase of cellular elements, unless hemorrhage has also occurred, by hypertension of the fluid, usually quite marked in the earlier stages, nearly always over 40 mm. and often over 100 mm. Labyrinthine disturbances are revealed by reactions to galvanism, rotation and douching tests. Marked amnesia is usually present, even after the confused state ceases. It is, however, an error to believe that the recollections which disappear are the more recent ones. In many cases the memories lost are those of childhood, while those of the war persist. The law of Ribot is not general.

HYSTERICAL COMPLICATIONS OF COMMOTION

The symptoms of these patients are not removable by psychotherapy, but it must not be forgotten that the suggestibility of a man in this state is highly increased and many hysterical symptoms do show themselves in patients suffering from cerebral commotion. The removal of these symptoms by psychotherapy is no proof that a man has not been shocked. Thus in one case after forty-eight hours of coma there was contracture of the lower limbs and complete mutism, although the former was made to disappear in a few minutes by suggestion, and the patient walked. Now, although the mutism disappeared during a dream in the night, and a contracture of the upper limbs ensued after lumbar puncture, and a hysterical chorea developed a month later, and although this man was able to return to the front soon after, yet a diagnosis of hysteria, though manifest, was insufficient, for rachiocentesis proved that there had been a cerebral commotion, for the fluid was distinctly hemorrhagic.

Trauma or diseases which interfere with the activity of the cerebrum decidedly increase suggestibility.

Digitized by Google

Again a stay in a hospital furnishes suggestions toward hysterical accidents. Hence, neither the presence nor the rapid cure of a hysterical accident excludes a diagnosis of commotion.

NECESSITY OF DIFFERENTIATING HYPEREMOTIVE STATES WITH ANXIETY

Patients in this condition are often mistakenly labeled hysterical. Their condition is really quite different. Such patients are far from suggestible. It is difficult to obtain access to them, so absorbed are they in struggling against their anxiety. They often show tachycardia, vasomotor disturbances, excessive sweating, nearly always insomnia, often with nightmares. Their appetite is poor or they bolt their food. They are disinclined to smoke or do so feverishly and rapidly. They are restless. These symptoms are aggravated by bombardment, by contrarieties. The patient rarely shows any focal symptoms such as paralysis, contracture, mutism, deafness, vomiting. True convulsive attacks do not even occur in these patients, although an emotional crisis might be mistaken for one by a careless observer. These patients are not amenable to suggestion, although they are amenable to a rational psychotherapy that penetrates the mechanism of their disturbance.

HYSTERIA IN THE WOUNDED

When there is a wound, and more especially one which has implicated a nerve, the attention of the clinician may be diverted from a psychogenetic interpretation of a case of war hysteria. This condition is very common, although it has been so frequently overlooked as to lead to the assertion that hysteria does not occur in the wounded. On the contrary, there have been thousands of wounded men in whom a hysterical syndrome has grafted itself on the wounded part and has become so fixed as to require an intensive psychotherapy for its removal. The following examples and illustrations make this fact plain:

Digitized by Google

A soldier received a wound in the upper arm which resulted in the severing of the musculo spiral nerve. Suture was done. After the wound healed the man was sent back to his farm for a year with instructions to work. This he did, and returned for inspection with a dropped wrist and unable to extend the fingers. Examination showed that all the extensor muscles responded both to faradism and to tapping. However, no voluntary movement whatever could be accomplished by the patient, so that the chief of the service believed that stretching of ligaments and tendons accounted in large part for the man's incapacity. I saw no evidence of this, and immediately began to reeducate the movement by persuasion, with the result that in less than five minutes complete extension of both wrist and fingers could be accomplished by the patient, who was not backward in showing his joy over his rapid recovery. The mechanism in this case was very simple. Its foundation was the man's belief that because the nerve which raised the hand and fingers had been cut, it was impossible to make these movements; accordingly he had never really tried. With regard to these movements he had therefore not exercised the dynamic volition which the psychotherapy compelled him to do.

In another case the mechanism was one of pure suggestion, as the wound which existed did not directly affect the extensors, for it was in the flexor region (Figs. 1 and 2).

Still farther removed from the first mechanism is the next case; here the wound was in the metacarpus. It is possible that reflex inhibitions were responsible in the first place for part of this patient's inability, but the complete immobility was only psychogenetic. (Figs. 3 and 4).

Similarly it is very doubtful if anything other than a psychogenetic mechanism was responsible for the disability of the arm in a man who presented only a flesh wound of the shoulder (Figs. 5 and 6).

Digitized by Google

It will be instructive to compare these cases with one of those reported to the American Medical Association in 1912 in my paper on "Modern Treatment of Hysteria." In the case mentioned, in spite of an atrophy of the hand produced by a hematomyelia causing a great diminution of power, the patient was enabled to use the hand sufficiently to work as a mechanic after one period of reeducative persuasion.

The leading differential symptoms are that in a nerve wound there is a disappearance or at least great diminution of reflexes; also localized muscular hypotonia and atrophy, as against slight general diminution of volume of the limb which may sometimes occur from disuse during a hysterical paralysis. The modification of the electrical reactions is always present when a motor nerve is wounded, although these are not noticeable until ten days after the accident. The localization of the symptoms to the territory of the peripheral nerve wounded cannot, of course, be reproduced by suggestion. If the symptoms extend beyond this territory (a frequent occurrence), the additional symptoms, not having the above mentioned characters, can be made to disappear by persuasion.

REFLEX PARALYSIS

However, a diminution of power in the movements of a limb may result exceptionally in consequence of a wound that does not implicate directly the nerves supplying the muscles concerned and without the participation of a psychogenetic mechanism. This is believed to be in consequence of afferent impulses from a wound of another nerve or even of the connective tissues, which afferent impulses determine reflexly an inhibition or excitation more particularly of the vasomotor system. This usually leads to an inadequate blood supply of the muscles and poor nutrition, accompanied by chilling, which sign enables us to suspect the condition. Other symptoms are: alteration of

Digitized by Google

tonus, alteration of reflectivity, especially hyperreflectivity of reflexes and of muscles themselves to tapping, a slowing of the muscular contractions on electrical stimulation with anticipation of the tetanic curve, localized muscular atrophy not accompanied by reaction of degeneration, tremor on attempted use, diminution of the capillo-arteriole blood pressure, diminution of the oscillations of the arterial wall, cyanosis, osteoporosis, hyperesthesia of the muscles and nerve trunks.

The disturbances really present are usually enormously exaggerated by the suggestion brought by their presence. It is almost the rule to find the limb completely immobile and to be able to restore most of the movements by intensive psychotherapy. In some cases the morbid physical reactions gradually cease, but in others a tendency to rapid chilling of the limb and a diminution of dexterity has persisted for years.

HYSTERICAL ALGIA

More difficult to correctly diagnose, and above all to treat, are those cases of trauma without external wound or visible bruise, when the question is one of sprain of ligament or tendon. Even after the initial damage is healed the patient may continue to hold the part in an awkward position and complain of pain when any movement is attempted.

The same condition obtains even when there has been no trauma, in cases that have originated from a local neuritis, myostitis, or aponeurotic fibrositis, arthritis, tenosynovitis, resulting from chill, toxicosis, or perhaps from the assumption of a faulty attitude. Not only does the faulty position interfere with the function of the limb affected, but it disturbs the whole locomotory balance, as well as the patient's mental attitude toward his functions. We know that, as soon as a normal attitude is given to the part, the pain soon disappears. The problem then is to so influence the patient as to compel him to perform movements that

Digitized by Google

restore the limb segments to their proper relation. This has to be done by the devices of psychotherapeutic art. It is not necessary to conceal this in complicated mechanical apparatus, as more direct methods have proved the best. It is all dependent on the skill of the therapeutist.[1]

An algia is merely a phobia against pain. The degree of the pain does not affect the question. What is important is the patient's mental attitude, for functions may be performed in spite of most intense pain, and in certain cases when pain should be quite negligible it may lead to complete incapacity. The commonest of the algias during the war has been hysterical sciatica, but pseudolumbalgia and pseudogastralgia have also been common.

The suggestion that arouses the psychologic symptoms may be derived not from injury, but from disease. Such cases have been quite common during the war. The most frequent of them, perhaps, are those in which the digestion is concerned. The foundation has been a real breakdown of the dealing with the food in consequence of fatigue, irregular and hurried eating, and the taking of excessive quantities of food in conjunction with severe exertion. A man who is hospitalized for this cause has his attention strongly directed to his stomach, more particularly when he falls into the hands of a certain type of gastro-enterologist. The massive and repeated suggestions derived from his treatment furnish the fulcrum with which he very easily constructs hysterical gastropathy.

In some cases the only symptom of this is persistent vomiting. In these cases, however, there is little loss of appetite, and scarcely any falling off in weight, while pain is absent in many cases.

The circulation may be troubled by paroxysmal attacks of tachycardia. These are usually provoked by the arousal of emotions in the soldier, more particu-

1. See Case of Knee Contracture, J. A. M. A., 1912.

Digitized by Google

larly of those which have occasioned his removal from the line. The attack does not occur with the suddenness of a true paroxysmal tachycardia. There is not the sensation of cardiac shock, nor does the feeling of impending dissolution occur. The attack may last only a few minutes, but it may be several hours before the heart beat returns to normal, and it may last some days, although rarely. In some cases there are convulsive attacks in these patients. These are not necessarily hysterical,[2] however, but may be a direct reaction of the distressing emotion that occasions the tachycardia. They are, however, psychogenetic in their manner of provocation.

The so-called effort syndrome is sometimes purely hysterical in mechanism. The patient believes that his heart is damaged; the least increase of demands on it creates apprehension; the apprehension causes an emotional contraction both of heart and blood vessels with an increase of rate, which is felt and adds to the apprehension, and sometimes produces a rise of blood pressure, which further increases the cardiac strain. The success of treatment is proportional to the access the physician gains to the initial cause, which is the patient's apprehension. The progressive exercises which have been utilized are merely efficacious in proportion to the suggestive influence on the patients.

Tachypnea may be the principal manifestation. In these hysterical cases it is accompanied always by a general agitation excessive in degree, the patient complaining loudly, and marching up and down. There is no proportionate increase of pulse rate, but the patient seeks to remain in bed, and begins to gasp on the least movement. This is a frequent sequence to poisoning by gas, more especially of the irritating kind. It is sometimes difficult to differentiate the part played by respiratory damage from that of psychogenetic order.

2. Compare forthcoming book, Neurological Disorders of Warfare, for discussion of mechanism of these.

Digitized by Google

Hysterical sequences are, however, common. These complications are very easily removed by appropriate psychotherapy.

Incontinence of urine has been not uncommon. It is either the relapse of a former adolescent incontinence, or arises anew during warfare. It has often arisen in consequence of a mild cystitis, or in consequence of prolonged fatigue. It is very easily removed by firm psychotherapeutic measures.

Convulsive attacks have been common but have been cured by isolation and by torpillage, as well as by methods of persuasion.

A CORRECT INTERPRETATION OF THE MECHANISM OF WAR HYSTERIA IN RELATION TO ITS MANAGEMENT

The foregoing considerations will aid us to an understanding of the more simple mechanisms encountered in the hysteria of war. The case is less easy if there has been no wound or if the injury has been internal, as in a case following cerebral commotion. When hysteria is expressed frankly as a psychologic state the mechanism is apt to appear a little more complex. However, as in the cases that have a physical expression, it is merely a matter of a conditioning of the patient's reactions in conformity with the impression he has gained of the circumstance under which his disorder has arisen. It is purely a matter of comprehending his ideas and feelings. In modifying the patient's mental attitude, it is absolutely essential that the physician ascertain this attitude. To do so he must enter into the patient's own thoughts and feelings regarding his condition and that which has led to it. It is difficult for a soldier in the army to show to the army surgeon, who is of course an officer, just what he thinks and feels. The man's confidence is inhibited by the disciplinary habits he has acquired in the army. The medical officer who treats these patients must

Digitized by Google

therefore remove, as far as possible, his cloak of office, and persuade the man to regard him for the time being as his personal physician only. When this is done the man can more readily reveal himself, and show how he thinks and feels about his condition. The besetting fears that the patient has, the anxieties, the apprehension of difficulty, the feeling of unwillingness, the sentiment of loathing, the melancholic trends, will in this way all be unfolded to the physician who is willing to enter into the patient's thoughts, to understand and to sympathize.

This is only necessary when there has occurred considerable fixation of the patient's hysterical ideas. In the earlier stages their expression is already frank, as in the not infrequent dumfounded state in which men will often retire from bombardment. In this case an analysis is not necessary, for the man can be quickly shaken out of somnambulism and returned to duty. So apparent is this condition that the most important quality required by the doctor here is dynamic volition.

In the more subtle cases the doctor cannot successfully effect a cure unless he has an exact and clear knowledge of the psychopathic reaction common to these cases, and he also needs the power of penetrating into the patient's psyche—then he is in a position to lead the patient out of the woods into the light.

THERAPEUTIC TYPES

Patients suffering from psychogenetic troubles may be divided, from a therapeutic aspect, into two types of weighty practical import. They may be called the active, positive, hostile type, and the passive, negative, inert type. Obstinacy, however, is not a prerogative of the first type, for the inert patient may be exceedingly obstinate though not actively resistant.

1. The men of the first type are determined to retain their symptoms. They have found their disease a handy method of evading unpleasant duty, and they actively do everything possible to avoid the measures

Digitized by Google

needed for their cure. In some cases they resort to tricks and dodges to deceive the doctors and attendants. Or they bring an active opposition to bear on his efforts, and in some of the worst cases of all the patients consciously simulate or even openly rebel, in order to avoid the issue which they know in their hearts will lead to the disappearance of their symptoms, in consequence of which they will have to return to the duty which they desire at all costs to avoid.

The only way in which they can be cured is by effecting a psychologic change in them. The old biblical expression "to be born again" expresses the end to be aimed at. The morbid desire of the man to remain a patient must be substituted for by a new desire, an aspiration toward the life of duty. There are two ways of effecting this: one is by making the hospital and treatment uncomfortable or unpleasant, so that the man, finding that he has jumped out of the frying-pan into the fire, begins to change his point of view and decides to try to get well.

However, even when he reaches this stage it is not enough, for he is then only beginning what every pithiatric must go through, namely, the casting off of a morbid habit, whether it is the habit of a vicious attitude or the habit of disuse. It must not be forgotten that in some instances there are physical consequences of vicious attitudes and these have to be treated. Often the treatment of these is exceedingly disagreeable, quite apart from the good will of the patient.

2. The commoner type of patient exerts no active opposition to the treatment, but he is a man ruled by his automatism, and he finds it very difficult, or even impossible, to make, of his own initiative, the necessary effort to break the acquired habit. Whether it is one of omission or of morbid commission makes no difference in the principle required for its replacement by reeducation, for the patient has to be pushed by the doctor into doing that which he is unwilling to do for himself. Sometimes the motive required to rouse him

Digitized by Google

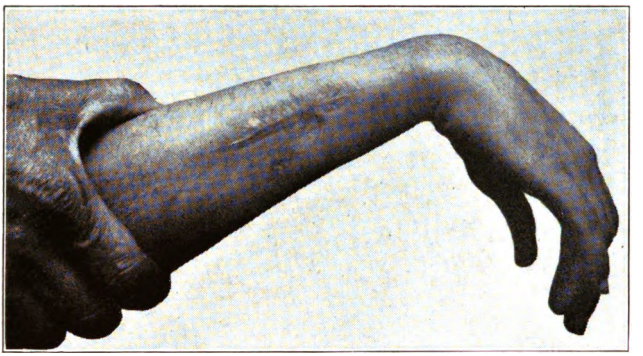

Fig. 1.—Musculospiral pseudoparalysis in a soldier whose wound is on the ulnar side of the forearm and who is endeavoring to extend the wrist. The palsy is pithiatric, most probably from imitation of the relatively common hand-drop seen in the hospitals by the patient while recovering from his wound. In a center of physiotherapy no benefit was obtained.

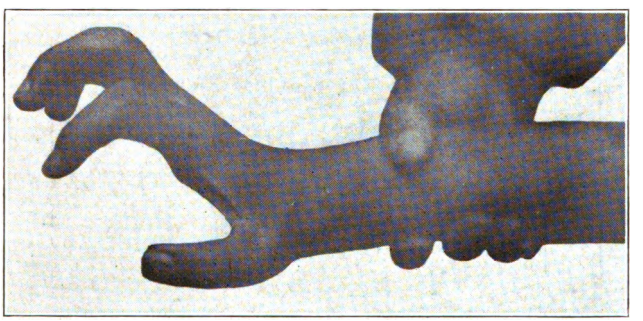

Fig. 2.—Musculospinal pseudoparalysis of the same arm as in Fig. 1. The condition of the patient half an hour after the beginning of psychotherapy by persuasion which taught him how and urged him to extend the wrist. The fingers are not yet extended, partly because of adhesions.

Digitized by Google

Fig. 3.—Wound of two metacarpophalangeal articulations. Before treatment the hand was completely motionless. Now after four months of treatment it can move in any direction. Vasomotor troubles existed also.

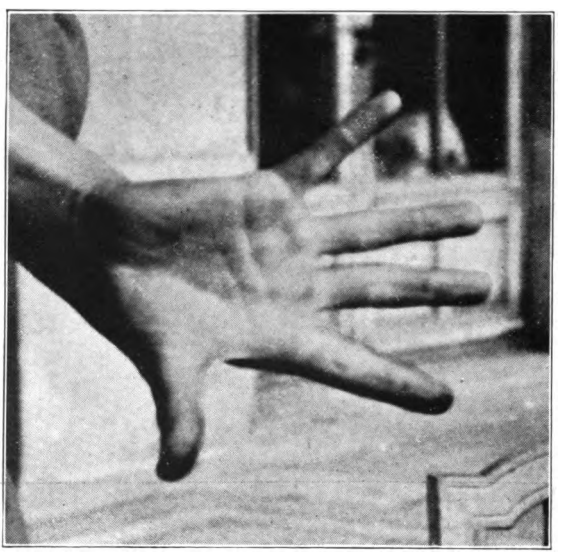

Fig. 4.—Opening the hand.

Digitized by Google

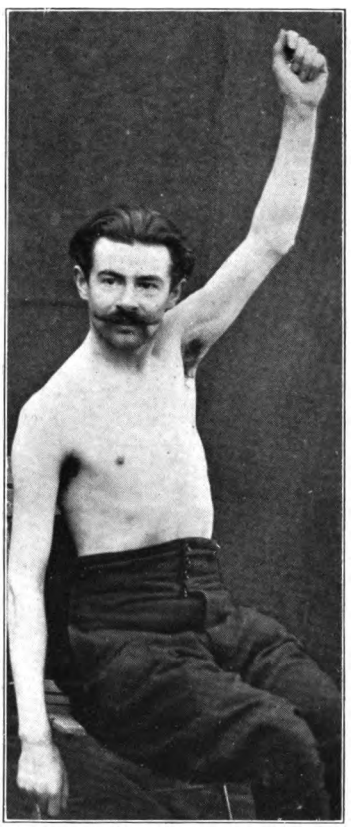

Fig. 5.—Complete monoplagia after wound of subspinal region. (Before treatment.)

Digitized by Google

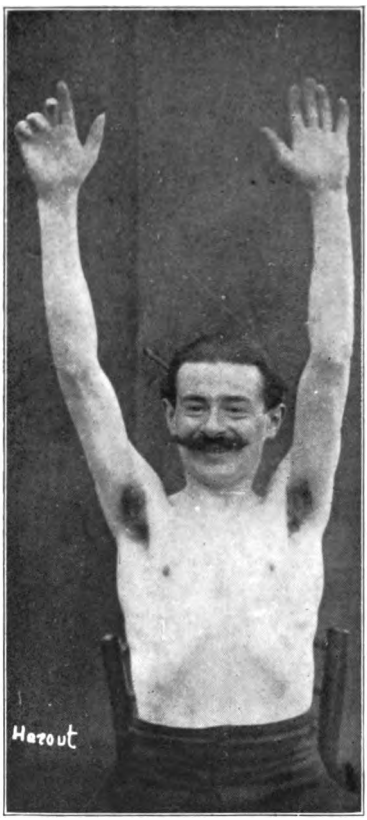

Fig. 6.—Complete monoplagia after wound of subspinal region. (After treatment.)

Digitized by Google

is, as in the case of the first type, dread of the unpleasant. More frequently, however, the method of side-tracking and indirect suggestion is the most rapid and effective in these cases, for in this way the man falls quickly and scarcely knowingly into another automatism, which a skilful doctor takes care shall be the natural automatism he formerly had.

As, however, influences against recovery are constantly being exercised against these patients, not only by thei rconcealed motives against a return to duty, but because of the hurtful insinuations on the part of comrades or relatives or perhaps, even worst of all, on account of a conventional opinion that a neurotic soldier is incapable of further duty, the method of therapeutics, which has as its principal weapon dislike of discomfort, inevitably produces discontent and even rebellion, latent if not overt. This inevitably leads eventually not only to many individual failures but even to a breakdown of the organization which relies on this method as its sheet-anchor. Accordingly, instead of the motive of repulsion being used, it is infinitely better to employ that of attraction: the former is a derivative of the fear instinct, biologically a negative chemiotaxis; the latter is an integral element of the positive motive power of existence.

In the process of the inculcation of motive, every teacher of the young since Fröbel has learned how vastly superior to the method of intimidation is the method of provoking interest. Even the method of repression is only of use as a means of breaking the chain of attention elsewhere than to the point desired. It is only when the subject becomes actively interested in what is being done that progress is made. So, in patients in whom psychologic trends are the root of their disorder, the dragooning is only the first step. We are back to the old saying, "The fear of the Lord is the beginning of wisdom." Some people forget that it should not be made the end. Hence, in the case of

Digitized by Google

BOSTON MEDICAL
SEP 24
LIBRARY

a pithiatric, he cannot be considered as safely restored until an active motive of desire to return to duty is substituted for his motive of desire to avoid discomfort. In some cases this is merely a flabby acquiescence in a general atmosphere. Such men are always liable to mental contagion, and their future, as their past, is dependent on their environment. However, even in men of this kind, a course of training can endow them with an astonishing obstinacy in well-doing. Very appropriate, in this connection, is the profound wisdom uttered by Jeremy Taylor when he saw the criminal led to the scaffold: "There, but for the grace of God, goes Jeremy Taylor." In the situation we are considering the grace of God is represented by the motivation derived by the patient from a skilfully devised environment. Those who have dealt with markedly hysterical cases know how faithful these patients become to a service which they may adopt. This is by virtue of their very defect; namely, the accentuation of their automatism, so that patients of poor judgment and highly suggestible may be moulded into very useful members of their group, provided they are not asked to step out of their regular groove. Even men who are intellectually quite low in type may be converted into valuable soldiers if this principle is utilized.

In persons of greater intellectual complexity, and particularly when the affectivity requires management, the appeal will have to be less simple, and the patient's own mental resources must be more largely deployed. To do this the physician must have a more extensive knowledge of the patient's personality than in the former cases. This is particularly true in the case of the actively resistant type of pithiatric. We have the right to believe there is a key to every one's heart. It is the psychotherapeutist's business to find it, and when he does find it, to tax his ingenuity to open the door of an active and useful life to his patient. These men are worth saving, for the very qualities that enable

Digitized by Google

them to resist actively will be of the greatest value to them as combatants. It is all a question of motive and desire. The eugenist who excited so much commotion at the international conference in London when he said that he would rather be the son of an efficient burglar than of an incompetent bishop, had this principle in mind. The finding of effective motives and the helping of the patient to utilize them is the aim of the psychotherapeutist, and his success in doing so is the measure of his utility to the army, and inded to humanity.

It must be clearly understood that when the highly theoretical and often confused concepts regarding hysteria espoused by some neurologists were allowed to influence practice in the army, there was an enormous overcharging of the medical services with functional disturbances of this kind. For these concepts entail a consumption of time out of all proportion to what is at the disposal of the medical officer in time of war. Besides which, they are therapeutically incomparably less fruitful than the concept of hysteria which we owe to Babinski, which has enabled neurologists of France to cure, generally within a few days, at least 70 per cent. of the hystericals from the front, and later enabled those neurologists operating in the army zone to send back to the front almost 100 per cent. of the functional disturbances of this nature. Even the most prejudiced will be compelled by the fact of this war to now acknowledge the utility, even if they do not care to face the truth, of the interpretation that traces the induction of hysterical symptoms to suggestion and which demonstrates that the so-called traumatic neurosis differs in no way from pithiatism.

Thus, the whole treatment of hysterical manifestations depends on one principle, namely, the replacement of the patient's morbid mental attitude by a normal one. It is a reconditioning, a substitution, a reeducation to which the patient has to be persuaded.

Digitized by Google

METHODS EMPLOYED

The means of persuasion are innumerable. Those most successful in the hands of one therapeutist should not necessarily be employed by another. The choice depends far less on the nature of the hysterical symptom, or even on the nature of the patient's make-up, than on the temperament of the therapeutist himself. Some men are more successful when they employ methods which in reality are pure suggestion. Others are more successful if they use methods which make the patients suffer. Others again cure more patients when they are permitted to elaborate a systematized reeducation of the patient.

SUGGESTION

The methods that are scarcely more than direct affirmation and suggestion are only of utility during the whirl of the dressing station at the front, at a time when the man's belief that he is justified in reporting sick is not at all firm. The diagnosis can be made swiftly and easily. To restore the patient's military capacity it suffices to assure him confidently that his trouble has disappeared and that there is no reason for its return.

TORPILLAGE

The removal of hysterical symptoms by the infliction of suffering is applicable to perseverators and simulators rather than to the ordinary hysterical patients. It is a method most readily employed by those physicians who have not the patience and the spirit of organization required for more systematic treatment. It is, of course, a very rapid method, saving a great deal of time for the doctor, and providing a great economy of man power, in that a patient treated in this way can be restored to the service in less than a month, whereas a patient in whose case the more moderate method of influence and reeducation are employed required four or five months to become fit for service.

Digitized by Google

The severe and painful methods of treatment, however, are greatly restricted in utility unless they are employed throughout the army, unless the assurance that they will be employed is known to the soldiers, and unless their use is sustained by public opinion. When the chance of evading this treatment exists either because the patients know that the doctor dare not push it or on account of fear of interference by political appeal, the treatment loses its authority and becomes a very painful ordeal for the doctor using it. Besides not every man has the tenacity, the courage and the skill required for its utilization. Mere relentlessness does not suffice. The doctor must know when to cease the painful stimulation and invoke the patient's own will in the removal of his disability, for intemperate zeal and bungling brutality inevitably produce a spirit of vindictiveness in those subjected to the treatment which has a detrimental effect on the neurologic services in general.

REEDUCATION

Thus, unless in thoroughly successful hands, torpillage should never be attempted. Accordingly, it is necessary to have recourse to the method of gradual reeducation. The principle utilized to effect the metamorphosis of the patient's mental attitude by this procedure is that of the building up of hopeful expectancy by the giving of examples of cure, and by other means calculated to create an atmosphere of confidence.

An organization which effected this purpose with striking success was that of the Seventh Region of the French army. It consisted of a central admission and reception center where the patients were first examined, but where they also came in contact from the first with a number of soldiers who had been cured of obstinate and long continued hysteria of physical allure, such as paralysis, contracture camptorachia, stammering, tremor, etc.

Digitized by Google

Treatment, however, was not conducted in this center; but the patients, after a period of observation and infiltration by hopefulness, were removed to Salins. There, after another period of collective suggestion in the neighborhood of cured patients, treatment by the rapid method was attempted. It was nearly always successful at the interview, but care is taken that the improvement obtained then is maintained by vigilant observation for two or three hours, together with almost continuous exercises during that period, under supervision of the instructors who have been trained by the physician for this purpose. After some days or weeks of fixation of a cure, patients are then transferred to a training battalion under officers acquainted with the principles of the treatment and the object to be sought in the fixation of the results. The procedure is described in detail in my forthcoming book, in which what occurs in the therapeutic interview is related word for word, and numerous examples are presented.

Relapses of cured hystericals will occur unless either the men are imbued with intense military fervor, or unless those who are not capable of this are met on the lines of communication, at the depot, and in the regiment, by a firmness of dealing gained from instruction of the officers regarding the nature of the condition and the manner of its prevention in cured soldiers. This organization was accomplished eventually in most of the French army corps but had not been organized in the American Expeditionary Forces when the armistice ended the fighting.

ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. WEISENBURG AND WILLIAMS

Dr. Robert McGregor, Saginaw, Mich.: There does not seem to be any essential difference between war neuroses and the neuroses of common life. Whatever distinctions there are lie in the severe character of the causal conditions, the intensity of the symptoms and the facility with which the symptoms interchange between those of anxiety states and those of hysteria. I had the good fortune to see many of

Digitized by Google

those cases in military hospitals in England and France and also in civilian hospitals in London among women and children as a result of air raids. I believe the problem of treatment is psychologic. At the same time one should not forget that many organic conditions are bound to underlie those manifestations, and the victim of severe shock must always remain the potential epileptic.

Dr. Charles A. Rosewater, Newark, N. J.: While on duty at General Hospital No. 30 at Plattsburg, I saw a case which presented features of unusual interest, and demonstrated, as stated by Dr. Weisenburg, that hysterical manifestations are induced by suggestion and can be cured by suggestion, even in cases of long standing. For about ten years a soldier had contractures of the right toes, with rigidity of the right ankle and partial rigidity of the right knee. He limped perceptibly, walked on the outside of the foot. He was unable to rest his weight on the foot. There was almost complete anesthesia of the entire leg. It appears that while working as a carpenter, his foot was crushed in an accident. Amputation of the toes was advised, but the patient declined and was treated by his physician for several months. For weeks the foot was kept in an elevated position, followed by a period during which crutches and later a cane were used. When finally the dressings were discontinued, the doctor is said to have declared, "I saved your foot, but you will never be able to use it." In response to this suggestion, the patient never used his foot. It is difficult to understand how the soldier was considered fit for overseas duty, but, nevertheless, he was sent to France. While drilling he fell, and being unable to keep up with his company, was returned to this country with the diagnosis of "hysterical paralysis with contractures." Learning from him that he was a singer, I reminded him that if he held back the tones he could not sing, and explained to him that his inability to use the foot was entirely due to the fact that he was holding back the impulse necessary to move the muscles. I assured him that he was perfectly able to move the foot which was a trifle weak from disuse. He reacted favorably to the reeducation seances, and after a few days was completely cured, and walked as he did before the accident ten years ago.

Dr. W. H. Hicks, Newark, N. J.: As a member of the advisory examining board of the state of New Jersey I saw many cases of psychoneurosis and I would like to ask Dr. Weisenburg if the psychoneurosis which occurred in many cases was due to a fundamental law of self-protection, a psychic inhibition, the best way for the patient to escape danger and founded on fear; if such was the case, how could one determine a real psychoneurosis that produced a monoplegia from a malingerer who had sufficient willpower to simulate monoplegia?

Digitized by Google

Dr. Lewis J. Pollock, Chicago: The policy adopted by the A. E. F. in the treatment of the psychoneuroses was such as to centralize the patients and to treat them as soon after the development of the condition as possible. For that reason relatively few cases were seen in base hospitals. However, during the early months of the active participation of the A. E. F. in this war it became necessary for a considerable number of these patients to be sent through base hospitals. I had the opportunity of observing about 350 cases of psychoneuroses developing at the front. Although agreeing with Schwab that the history of neuropathic hereditary taint did not frequently make its appearance, it was noted in a large percentage of cases that the patients themselves gave a history of having a certain type of make-up which we are accustomed to speak of as unstable. By this I mean that the individual had always been oversensitive, hyperimpressionable, easily upset, inclined to overscrupulousness or diffidence and embarrassment. Frequently he was the subject of various fears, as fear of lightning, high places, at times fear of loss of consciousness and rarely death or insanity. Though the fundamental features of the war neuroses are the same as those of civil life, the picture in its entirety differs in many respects in the cases observed in France from the condition met with in civil practice. This difference is probably due to the numerous conditions tending to produce the neurosis and which occur consecutively. From the time a man is inducted into the army until his return to civil life he is the potential possessor of a neurosis. Conditions in training camps, on transports, in rest camps, in cantonments, in France awaiting active service, in battle and in base hospitals on the way back from the line, all contribute to the formation of a neurosis. It is, therefore, rare to meet with only one type of a neurosis in a single individual. If a conversion hysteria develops in a soldier as the result of any situation which might be present, the rapid disappearance of the symptoms following the many defective methods of treatment does not mean that the man is well. Most frequently when the hysterical symptoms disappeared an anxiety neurosis was found to be present, the treatment of which was essentially a different thing and one marked by slow recovery and stubbornness. It is surprising to me to note that Dr. Weisenburg reports so many cases showing the stigmata of hysteria, as aphonia, paralyses, etc. Inasmuch as it was the policy of the neurologists in France to send to the United States only such cases as were constitutional and inasmuch as the hysterical stigmata, whether occurring in constitutional cases or not, were so easily cured, it occurs to me to inquire as to whether these stigmata did not redevelop during their journey homeward. I do not believe that the desire to avoid duty at the front contributed largely to either the formation of the neurosis or its incurability. It was not the

Digitized by Google

fact that the individuals consciously wished to avoid service, but their character formation was such that they were unable to successfully cope with the situation and although frequently consciously wishing to "carry on," unconsciously the neurosis developed as a means of escape from a situation which produced a conflict between the man's social and moral desire and his incapability. It was only in this manner that the desire to avoid service contributed to the formation of the neuroses and not in the sense of disloyalty or cowardice.

In reference to Dr. Williams' paper, it is an interesting fact that the so-called physiopathic reflex nervous disturbances have to my knowledge been relatively rare in the United States army. Among the several thousand wounds of the extremities which I have seen, and included among which are 800 peripheral nerve lesions, no more than six such conditions can be recalled by me. I would like to ask if it has not been the experience of the French that these disturbances occur later in the course of the various wounds, and that sufficient time had probably not elapsed for their appearance among our wounded?

Dr. Theodore H. Weisenburg, Philadelphia: The differential diagnosis between hysteria and malingering is not easy, and in Plattsburg it was one of the most difficult of the questions that we had to decide. The reason for this is evident because hysteria itself is a motive disease, whereas in malingering the whole basis is that of a desire to deceive. I recall one case that gave us particular trouble. This man had a bent back. He developed this condition seventeen days after he entered the service. After a year of treatment in various hospitals, he was finally made to straighten up in two or three minutes, which to some of us proved that he was a malingerer. Yet other careful neurologists had found such disturbances in him as hemianesthesia. Therefore, it was difficult for us to label him finally as a malingerer. Dr. Pollock's discussion is very interesting and intelligent. His experience was different from mine in the sense that he dealt with neuroses very soon after combat, whereas the patients we had in Plattsburg approached peace conditions. As he stated, it was the policy of the War Department to return to the United States, at least in the beginning, only the constitutional cases. Therefore it is only natural that there should be in these cases a great many with a psychopathic history. Regarding Dr. Pollock's question as to whether or not the symptoms persisted from the beginning, for it was his thought that there might have been intermissions, this might have been the case, but careful histories showed that in a number of instances the symptoms persisted from the beginning. Undoubtedly the fact that the patients knew that they would not be returned to the front had a beneficial influence in their cure and this was shown in the armistice, for while this did

Digitized by Google

not bring about any great change and there were no spontaneous cures, yet our work became very much easier.

Dr. Tom A. Williams, Washington, D. C.: I agree that it is very difficult to detect malingering. It is done by contingent circumstances, rarely by tests. It is important to study the mechanism of war hysteria. It is not always caused by fear of death but often by disgust of conditions. The anxiety cases must by no means be treated by the same methods as the hysterical cases. They cannot be modified by suggestion. With regard to deafness or aphonia, many cases originate in actual physical trauma. The actual effects may disappear in a few hours, but the man thinks he is still deaf or dumb until persuaded that he is not. The same with a bent back. He will not straighten up because he is convinced it will give him pain as it did on the occasion of the injury. The reflex paralyses are rare. But there exist cases of atrophy in a limb after operation on the opposite limb. The views I hold with reference to these matters are not from partial observation. I have seen hundreds of cases, both early and late, in French, British and American armies, and I see nothing inconsistent between war syndromes and those we previously knew of hysteria in peace, neither their mechanism nor their manner of response to psychotherapy differ in principle from what we previously knew. The war, however, has given us a better way of managing those methods by which we apply those principles which have long been known to us. It has also diffused the knowledge of what hysteria really is.

Digitized by Google

THE DUAL PERSONALITY OF C. A. M.

THE ACCOUNT OF AN AMNESTIC FUGUE

EDWARD E. MAYER, A.M., M.D.
PITTSBURGH

Lapses of memory are commonplace incidents. They may deal with past or present experiences, be transitory or continuous, organic or functional. The episodic amnesiae of the epilepsies, the lost and confused memories of the toxemias and the infectious diseases, the amnesiae of the senile, and those resulting from trauma, especially concussion, are familiar varieties. This paper will deal only with the mechanism of psychogentic types, the narrative of C. A. M. being utilized to bring out our point of view in respect to certain phases of the psychopathology of amnesia.

We were at first puzzled in deciding what title to give to our recital of this history. This is not a new experience since behavior reactions are difficult to classify. The names applied to any given group of symptoms or the name of any mental mechanism conveys little information. It merely connotes the point of view of the observer or rather his method of approach to the given problem. Familiarity with classifications is too often considered to imply an understanding of the condition at hand.

The subject of this paper lost his memory of self and therefore his personal identity. As a consequence he disappeared and led, if his diary is to be believed, a most exciting two weeks filled with incidents which read like those of a cinema reel. If they were not true, they were the imaginations of a psychopathic person or were fabricated for the purpose of his being considered psychopathic, this inference entering

Digitized by Google

into the problem because of events which preceded his disappearance. We were apparently dealing with a dissociated personality. Our point of view had, however, to take into consideration the question of the simulation of such a state.

We are wont as physicians to ignore the inductive method of approach, and are prone to accept as facts whatever is offered to us by patients, interpreting them, however, only so far as they are borne out by the behavior reactions of the patient. The accumulated experiences of many observers has tended to fix for physicians certain standards of normal and abnormal, and deductions from them have, as a rule, not failed us; nevertheless, our facts rest on ideas rather than our ideas on facts. This patient compelled me to reexamine my own attitude in reference to the validity of double personalities in order to get my bearings and, in doing so, I was struck by the meager help furnished by the many writers on the subject. Most of them accept the facts as given by patient or observer and often fortify their acceptance of them in rather an inconvincing way. Perhaps this is because an inductive approach is impossible without more experience than the few double personality cases which fall to the lot of one observer. Perhaps it is because there is no absolute accordance in reference to the fundamental problems.

Before presenting the history of C. A. M., therefore, I wish to formulate a point of view concerning dual personality. All human beings have memories in their day dreams and in sleep as well as those which influence their conscious choice of activities and serve to motivate their daily life. That such memories originate from previous experiences, ignored or forgotten, we may also accept, I believe, as true. To what extent, suppressed emotions or instinctive trends influence these processes of memory in any given person it is impossible to say. The play of motives is a

Digitized by Google

difficult thread to unravel and yet underneath dreams and emotions, the path can often be followed if we find its beginnings. A person's experiences, so far as they are utilizable by him, are largely responsible for his actions. Habit formations and automatic responses show us that the decision is not always one of deliberate choice. In some types of reaction if the wished-for choice must be in conflict with reality, a compromise is attempted or a defense is erected in our psychic life which may be partially successful by short-circuiting vital energy (introversion) or through an inhibition of what is considered normal functionating or (according to Babinski's physiopathism) an overstimulation of physiologic responses. An amnesia may be regarded as a defense reaction, therefore, in the higher psychic spheres, the conscious mind shunting off in this way painful memories or other intense emotion and carrying with it all associated ideas. It is, therefore, always dynamopathic.

The use of the term personality connotes so far as I can see merely a person who is aware of being alive and conscious of his actvities. Some writers attempt to convey by this term a self who is striving for some goal in life. If this is the case, then of course an automatic state is not personality, a hypnotized subject has no personality, and accordingly some of the dissociated states which have been described under multiple personality are not such at all. A child is conscious of its endeavors to walk, but without enough understanding to be aware of any purpose in its striving. Yet a child has a personality. It has a disposition and personal characteristics, and is aware of the situations which arise around it though not entirely of its relationship to them. The awareness to situations is looked on by some as the test of personality. Awareness of certain experiences as contrasted with the awareness of another set of experience is the criterion on which is erected, by most

Digitized by Google

writers, a diagnosis of dual personality. This is in accordance with the opinion that most persons simply react to situations and cannot create them; that they are as Ribot says echoes, with no voice of their own. Yet, even though without initiative or originality, they have individual characteristics which make them personalities. The term, however, is hardly a psychologic one. It takes no account, for instance, of those reactions which explain a dynamic amnesia, problems of overactive instincts, atavistic trends, inner conflicts and defense reactions.

It is, nevertheless, convenient to have a word in use which does not require so precise a definition, to serve the purpose of associating the various cases of lost identity of self. If their mannerisms and mode of life also change, one has the right to speak of dual personality. Unless the changed self also creates and acts independently it is clearly a fragment of the old self—a dissociated state—and not a new personality. This is the criterion which some insist on.

In many narratives of dual personalities we find that the test of their occurrence depends not so much on a loss of self-identification as on a sudden and therefore startling loss of the memory of self. Altered characteristics, a change of vocation, even the lost memory of the previous experiences of a person would not be considered as indicating a dual personality unless the cleavage happened suddenly. But this is not enough. Somnambulists have no consciousness in their automatic states, yet we do not speak of them as double personalities because they are not conscious of their acts. To premise a double personality because of an altered plan of life with a loss of memory of the previous life, even though sudden in onset, would barely suffice. In fact, additional corroboration is offered in many of the reported instances through observations of changes in organic sensations. And more important still is the contrasting evidence of only

Digitized by Google

a partial loss of identity in most of the observations dealing with double personality, evidence of the honesty of the observations and of our acceptance of them as proof of the occasional occurrence of a complete loss of self.

It is hardly correct to speak of a dual personality in the case of one who is in a subconscious state, living the life of his submerged self, dealing with the past rather than the present. This is an acceptable opinion in regard to the production of a dissociated mental life. But dissociation, which is common, hardly constitutes a new personality, which is rare. A break in the unity and continuity of one's experiences must be not only sudden in origin and intense in quality, but must be accompanied by activating influences which force the person into new walks of life, in order to be considered as constituting a new personality. The breakdown of a mind leaving it a complete blank or with alternating changes in disposition, which characterize many of the examples in literature of double personality, do not answer to this view. They are, strictly speaking, really not double personalities any more than are any sudden differences in conduct or actions of a person except in so far as they have no memory of any alteration.

A lost memory of self is the fundamental basis of dual personality. All cases cited in medical literature have this in common. Differences, however, exist in respect to the retentiveness of present memories, changes in the memories of past experiences, the capacity to utilize past memories, accompanying changes in the sensory spheres, etc.

Memory being the only basis that we have for the identity of ourselves, we come to the conclusion that a lost memory of self is what is meant by lost personality, and a self which lives and acts without being personally identified with the former self is a dual personality.

Digitized by Google

THE STORY OF C. A. M.

This young man married into a family the members of which believed themselves above his station in life and were not averse in showing it. Gradually this zone of hostility became oppressive even to his wife and yet neither knew how to overcome it. His financial resources were meager and he had no means to keep his wife except at either his parent's home or hers. His parents' roof was impossible for her. After about a year of this, a method of escape seemed to offer itself and he seized it eagerly.

It was 1915, a year of wild gambling in stocks and its fascination and possibilities lured him on. Money was thus easy to make; he could in this way offer his wife a home of his own. And so the venture was made, it being understood by her that he had a new position with a larger salary and a chance for large commissions. Incidentally by leaving his position and becoming an automobile salesman, his opportunity for stock speculation was easier. He rented a large house. He bought his wife an automobile. He gave her on several occasions large sums of money, which he pretended to be his commissions. This happy life of hers lasted for a number of months. Then came his disappearance and her disillusionment.

It was on June 6, 1918, that he disappeared, and he was lost until he was picked up by the police at Asbury Park, N. J., June 23, 1918. During this interval many facts came out concerning him which had not been known. He was supposed to be in the employ of the B. Truck Company. This was not true. Some time in the spring, it was learned, a promoter from Philadelphia secured from him a few thousand dollars for a joint partnership in what was to have been a new fire extinguisher company. Although a contract was signed, this company never came into existence. In May, a few days after he realized that this money was lost, he went to Cleveland. From Cleveland he wrote to his wife, enclosed a key to their house, and a check, and told her that he was disgraced and would not come home. She, however, went to him and persuaded him to return to Pittsburgh. He told her that the money he had lost was money which he had earned and saved. It often happened that he would come to her with a check or money, accompanying it with the remark: "Here's what I earned today." These amounts totaled a fairly large sum, so that she enjoyed in these few months the use of considerable money. He had always been industrious. He enjoyed a good reputation. His habits were good. He never spent any money on himself. His energies seemed to be directed toward giving his wife what she craved. In fact, after he disappeared, his mother reproached her very severely for being the cause of everything because of her extravagant tastes.

Digitized by Google

His wife never saw anything peculiar about him. She, however, reports that he told her about "spells" which he had. The first one he spoke to her about was in December, 1917, or six months before he disappeared. She describes his account to her as follows: "I was in an elevator and for a minute I did not realize anything. The elevator boy told me that I looked very peculiar." At another time (in February, 1918), he came home and said: "When I was in my car I found myself in Carnegie. The last I remember was an hour earlier being in Pittsburgh." The day before he disappeared he went to Greensburg. He told me that he was going there with his employer to arrange for an agency for trucks. On his return he told me this story: "We were at the Rappe Hotel. I went to get a cigar. I didn't remember anything after that until I found myself in the dining room." After he disappeared I spoke about this trip to the family and his brother told me "Why, he wasn't in Greensburg with his employer at all. I was there with him and I didn't notice anything peculiar." After his marriage his wife received an inheritance (about $20,000) in securities. These were in her name and were supposed to be lying in a safety vault. After he disappeared it was found they were gone. Nor does any one know anything about them except that they had been negotiated by him between March and May of 1918. June 2, 1918, he bought a new automobile, offering a check for $2,500 for it, which check came back marked "No Such Funds." This was about the day he disappeared and the new automobile was returned to the company. Early in June he also gave a check for his June rent which came back unhonored. Such an occurrence was not new because in April he bought a dog for $150, which check was also returned in the same way. His wife, however, paid for the dog. There were also a number of other checks given out by him just before his disappearance which had to be made good.

He was noticed by the chief of police of Asbury Park sitting on a bench in the park. He seemed ill and therefore was spoken to but could not tell anything about himself. A locket was found on him which contained a picture of a woman and on the back of it were a name and address with the request to inform her in case there was any need. This was his wife's picture, her name and address. In this way he was returned to his home and came under my care. When I first saw him at his mother-in-law's home, he was sitting in a corner, listless and apathetic. His speech was slow and hesitating and consisted merely in answering: "I do not know" to everything, interspersed with the stereotyped remark: "I want to go to Honey. Where is Honey?" Honey was the name he used in speaking of his wife before his disappearance. She was sitting in the room when I first saw him. "Who is this woman," I asked. "I don't know,"

Digitized by Google

he answered. "She says she is my wife, but I want Honey. Where is she?"

On the next day his answers to the same question from her were: "You are like Honey. You are so good to me. Won't you take me to Honey?" And then with tears trickling down his face: "Won't you take me to her? She needs money. I want to see her. Oh, I wonder where she is. Who am I? Can't anybody tell me? Who am I?" My physical examination revealed none of the physical signs of hysteria.

THE PATIENT'S RETURN TO SELF

I had him removed from his home to a hospital in order to isolate him from his people. He brightened up at once. No symptoms of any psychosis were noticed. His amnesia was the only evidence of anything wrong. I made no effort for a few days to try to bring out hidden memories but employed my time in informing him of what a young man who was C. A. M. had done or was supposed to have done, and that he was supposed to be this young man. It was rather commonplace to call to his attention the fact that he had a picture in his pocket through which he was returned to the home where I found him, and that this picture was of the woman who was so good to him, who asserted that she was his wife and yet whom he did not remember as such. I, also, without any further comment called his attention to the fact that he was willing to accept her hospitality to a greater extent than seemed proper unless he did know her, since he had spent two nights at her residence, that this woman whom he did not recognize as his wife slept in the same room; that she helped him with his dressing and assumed in every way the obvious duties of his wife, and that he accepted these attentions from her. He may not recognize her as his wife but his mind accepted her as such. I had him go over with me the advertisements of some magazines, telling him that he must have done work before and that he could arrive in this way at something connected with his former life by noting what kind of advertisements interested him. He was very much interested in automobile advertisements, although, of course, this would not be unusual for any young man. I had him also try to guess in what city he would like to work and he developed a line of associations connected with Cleveland. Cleveland is the city to which he had fled for one day before his final disappearance. Cleveland was, also, the home of some automobile industries. I asked him to guess what he wished to do there. As a natural sequence came the voluntary offering from him that he would like to get connected with some automobile concern there and that it was such a pleasant place to live in. He guessed also that the woman who said she was his wife might be his Honey. I made no comment to him on his

Digitized by Google

sudden acceptance of the situation as it was and of dropping his quest for any other "Honey" the day after he entered the hospital, or of his willingness to start life over again in another city. More than this, after another day of this kind of conversation, my suggestion that his plans, of course, included Mrs. C. A. M. was answered in the affirmative. It was very easy after this to have him fuse his actual wife into the woman who had received and nursed him after his coming to Pittsburgh. Denials were made, however, even after he had assumed his normal life and returned to his mother-in-law's home of the incidents preceding his leaving Pittsburgh. He never acknowledged any memories of them. Because of the attitude of some of the family in reference to possible criminal charges, I was chary about pursuing this too far and did not resort to hypnotism in order to try to ascertain more of his inner life.

This recital sounds very uninteresting and would read manifestly as if C. A. M. had a fictitious amnesia. And, yet, there is more to it. We have been able to trace some of his life. In the first place, he did not ask for help at Asbury Park, but was found in the park manifestly ill, suffering from lack of food and giving evidence of having tramped a long distance. At the hospital a reporter elicited the following from him, according to the account in a local paper which I will quote:

The man appears to be less than 30 years of age, is neatly dressed, wears a soft white collar, is slender and rather tall. He carried with him a book in which he apparently had recorded many happenings. He dwelt on a story of his abuse by a man who had cared for him and later when the man found out his ward was "learning too much" decided to get rid of him and put him on a train. He tells us also a story of how the man beat him, although he did not know who the man was or where he lives. He has no marks of identification on his clothing but has a pocket knife bearing the initials C. A. M. and also a pocket edition of "Proverbs." He carried in his pockets two towels carefully folded with some soap. He told Detective Sergeant Broderick that he had a slight recollection of being on a ship and remembered that he would be lost forever if any lights were lighted during the night. His memory did not go back further than yesterday, when he says he walked into this city from the country. He came in contact with the police of some large city sometime ago and a detailed account was made in his note book.

Digitized by Google

The account of his experiences as written up in his note book follows: (The blank spaces are indecipherable words):

THE PATIENT'S DIARY

This book was found on me when I was taken to the police station. The name in the front was what I was told to be my name. The big fat man there said I looked like a boy friend by that name and since they didn't know my name I suppose I shall have to keep it. They told me I was taken to a hospital because I had nothing to eat and I had fainted. Oh, why! oh, why can't I remember who I am. I don't seem to know any one here. I can't remember one thing. I don't even know my age. I know my name is not Graverson and must be something else beginning with an M. because I have a ring on with the initials C. A. M. I am told at the hospital to keep this book and every night to record in it everything that happens and then if I ever do find out who I am or where I belong I'll know what has happened. Oh, God! that I had my memory. Please some one; please help me find out something. Everything seems black behind me. I can't remember anything about myself. The big surgeon says something awful happened to me or else I must have seen something terrible happen. He says that's why I have no memory of myself. I did feel badly for them when I was there. They tried so hard and I tried so hard to think. All I can remember is that somewhere I love (at least, they tell me that's the word) some one an awful lot, because in the hospital I kept calling for Honey all the time for the days I was there. Oh, how I wish I could find Honey. Please, please, dear Honey, won't you come to me? I can't remember you, so you must look for me. I'm here in New. . . . But they tell me to pray God for my memory. Well, then dear God, please give it to me. I'm going crazy in the state I'm in now. If I were blind, it would be the same sensation. But if only I knew who Honey was. Dear Honey, I really love you with all my heart and soul and so please, please come to me. I'm awfully lonesome and I'm cold and so hungry. I only get one meal a day and so I'm all cold and hungry tonight. If you do love me I'm going to wait until you find me. Please don't keep me waiting long because they say you'll come. If you don't I shall starve. No one here cares what becomes of me, but I do. I know I look as good in a looking glass as some rum I see on the streets and some . . . I know I should be. But Honey, what will I do soon? My clothes don't seem to be good and there's a tear in the sleeve. I can get another they tell me if I give money for it. What is money? Nothing but paper with something printed on them and little round things. What good are they? Why

Digitized by Google

will they give me clothes if I give them money. They say you must give a lot of it. I haven't any at all. I wonder what I'll do when this suit wears out. So Honey, won't you please come to me? This . . . is . . . What is . . . What does it mean? Oh, Honey, dear; please come to me now. Everything is black before me now. Please come to me I need you.

I don't even know when I wrote what's before. I wonder what happened; my writing is so awful at the top here.

It's been a long time since I've written in this book. It seems a year or more. It seems I have not written anything for so long I have forgotten a great deal. I shall write down what I do remember and leave the days absent 'cause I don't remember them. As near as I can recall I was in some small hotel down town and I took sick. I tried to keep up and tried but my legs were so weak I couldn't stand on them. Of all great joys I can now remember of going to school and learning to write. Can it be possible that my memory is returning. Well, at any rate, they took me to some hospital and I stayed there some weeks. Soon I got well and they let me out and gave me some silver. As I was walking along some street, a man came over and spoke to me and I answered him because I thought he must know me. I asked him. He said yes and I felt so happy. I thought that soon I would be home again wherever it was. When we got on a car in a tunnel we rode and rode for a long time. At last we got off and went into a house and up an elevator to the twenty-ninth floor where he said I would stay that night. He took me in and washed my hands and face and after waiting a while we sat down at a table and had something to eat. It wasn't much but it tasted good. At the table there were nine other people, all of them women and none of them dressed very well. They were all young girls but one, who said she was 95 years old. At the end of the meal the table was cleared and one very pretty girl got up on the table and took off a coat. After that she didn't have no more clothes. She jumped around for a while and then this man told me to go with her. I went and we walked a long way through a very dark hall to a small room. We went in and she locked the door. Then she laid down on the bed and asked me to do so too. I must have remembered something 'cause I knew that was wrong so I didn't do it. Then she fought with me and blood came out of my mouth. She hurt me 'cause I cried. Then after a while this man came and took me away from her. Then we walked through this long dark place again to another room. He asked me to sit down and drink some brown stuff. He drank some, so I did too. Then he asked me if I knew anything about an automobile and I said no, but he wouldn't believe me and we got on another car in a tunnel and rode along a river a long ways. It must have been late when we got

Digitized by Google

there 'cause there was no one around. He took me in a door and then down some dark steps and past a lot of horses to some big room where there were a lot of automobiles waiting. He took me over to one and said it was his and I would have to fix it. I told him I didn't know how and he hit me in the face and told me to get inside it. He hurt me worse than she did. He showed me something about it and told me to fix it. I couldn't but he made me use some tools as he told me. Then he made me stop and he went all through my clothes and put his hand in all my pockets. He took a lot of money from one of them. I didn't know I had any and I don't know where I got it. He then kissed me once, told me to put my clothes on again. I have so much to write I am getting tired but I must write it before he comes back again. After I got dressed we got on a car again and went to some place else and went to bed. I was awfully tired and went to sleep very soon. When I woke up I was alone so I got dressed and found the door locked. So I waited all day for him. He came back again at night and took me back to the house where we first were. We had a little to eat and then we went back to this room with the same girl. This time she cut my hand with a knife. Then she locked me in the room and I stayed there a long, long time. They didn't give me anything to eat. I got sick again so they took me out and gave me some dark brown stuff to drink. Then we went back to where the car was and he made me stay there for three days, and use some tools. They didn't give me much to eat and I had to sleep sitting up. After a while some other men came and found out and he was awfully mad at me. He hit me every time he spoke to me and once he put a sharp thing in my arm and it hurt for a long time. After the car was fixed six of us got into it and went to some place to eat. There was singing and lots of young girls there and here I got enough to eat. He made me drink some dark stuff which I liked a lot. Then he saw some other people he knew and we all went up some stairs to a room. The room was all lit up with big lights and then we drank some red stuff and these seven men and two girls talked together for a long time. When the lights went out we got in the car and went back to where we found it. Then we went back again to this house and they made me go in a dark room to sleep. I couldn't go to sleep so I stood up on one spot all night. All night long I heard them planning something, several times I heard them say five. The next day I didn't get out at all. I stayed inside and all the time kept thinking of that some one—Honey. Who is it? All I remember is that I cried all day for Honey to come to me and take me away. It's too bad to be this way 'cause I know that whoever Honey is I care a lot for and want. I have tried and tried and tried to remember who I am but I can't. I don't know anything and it's so hard, so I told this

Digitized by Google

man to try to find Honey for me and he said he knew who I was. He told me my name was Mason but said he would not let me go home. I wish I had a cannon, I would shoot him. He knows I want to go home but still he won't tell me where I live. So I'll have to stay with him until Honey comes. So that night we went out early and rode and rode and rode a long time. Then we came to a long row of houses and went down some steps into one. As soon as I got in I saw some funny looking men all talking. . . . I could not understand them. They took us to some small room where there were little beds. Here I saw about four men and two girls lying smoking from long pipes. Everything smelt so funny too. My pipe doesn't smell like that. They all seemed to like it. Then he hit me for not going into one of these beds, so I did and I smoked one of these pipes. They were fine but made me feel so funny. The last thing I remember was I got dizzy and I must have fallen asleep, for, as I woke up, I heard an awful cry and as soon as I could I looked over the side of the bed and this funny looking man was lying on the floor and he was bleeding from the back. I tried to get up but he hit me with something and that's all I remember. When I woke up the next time I could get up and I couldn't see any one around so I took a walk through one of the halls. Everywhere I went I saw men and a few women asleep in these beds. Off in the corner I saw one woman with nothing on and her head all bandaged up. When she saw me she threw some cover over her body and I went over to talk to her. She said some one hit her and took all her clothing. Then all of a sudden I seemed to know her but I couldn't recall who she was. She said she didn't know me but I still think she was my Honey that I remember by name. I sat there and looked and looked at her but I could not remember who she was. While I was there she told me that I was in the underground horror home and that I could not get out, but just then this man came in and grabbed me and took me away. I asked him to take her too but he just slapped me. We went upstairs and got into the car and went to the first place we went when I first met him. This night he made me sleep with him in some big room where there were lots of funny animals and I did not sleep all night. The next morning he told me I know too much and he was going to take me away. So that day he put something over my eyes and when he took it off I was on some ship. It was late and I was tired so all I could see was water and . . . straight line of lights. When I woke up he told me not to turn on any lights because we might have to swim . . . he said there was an enemy ship close at hand and they were after us. He then took off my clothing and tore . . . pieces of paper. As I was drinking some dark stuff he put something up to my nose and I went to sleep. It was fine. When I woke up the next

Digitized by Google

time we were going off this ship and then we got on another for a short ways. Then we walked for a long ways and he . . . out a paper for me and made me write Clarence Andrew Mason at the bottom. He then told me to go through a door where there were a lot of men and give them this paper. He wrote down on a piece of paper this number and told me to give it to this . . . inside when he asked for this. He then gave me another sheet of paper with a name on it and said to also give them it. He. . . . I waited to go on this ship. He said if I didn't do it he would kill me . . . I got . . . waited a long time and thinking they were getting me some clothes and I was taken on to the ship. [After] Three hours some man came and took a card from me and my ring, my name again and they gave me some money. There were two pieces of paper money and some silver. These men made me get down into some small hole and use a hammer on some big thing. I don't know how long I was there but there were an awful lot of men there and an officer seemed to be closing the . . . thing. The last day I was there I made up my mind to go away and see if I couldn't find some one who would help me to get home. I tried to get out but couldn't; they wouldn't let me, so I went back again, but the men there said I would get a pass to get out the next day so I went back and slept that night. This man also gave me some dark brown stuff and it certainly tasted good. . . . I cried and cried for my Honey, but still she didn't come. I then got a pass and put on all my own clothes again and got out this sheet of paper. I spoke to some man and told him I did not know who I was and he said to give him all my money and he would buy me a ticket on a train to some town. I gave it to him and he put me on some train and told me not to get off until the train stopped running. I rode all night and got here very early this morning. He also told me to go to the largest hotel and ask for a room. He told me what to do, so here I am tonight. I don't like the looks of this town. It reminds me of the place where this horrible man lives and I do not want to go to that death horror home again. I'm so hungry too. I haven't had anything all day to eat. Tomorrow I'm going to start walking. I don't care where I go only I'm going to walk until I get to some one who will try to get me home. I don't care whether I get home or not so long as I find my Honey. I know she will keep me 'cause I love her so much. I just met a man on the street who found a picture of a girl in one of my small pockets and he told me to go to Pittsburgh because of something that was written on the back of the picture. I am going to carry that picture until I find Honey. She may be here but I do not remember this picture.

I have just been reading what went before this and as nearly as I can remember this man found me again. I know

Digitized by Google

the hole in the book is where he tried to kill me but he did not 'cause I got away: I am now walking. I don't know where and I've been crying every day for my Honey. Every one I tried to talk to today I . . . I can't talk now so I don't care now what happens to me. I can't talk and I don't know what I am so why should I want to live. I found some money today so I'm going to walk until thats all gone and then I'm going to kill myself. I wonder why I can't talk. I could before but I suppose its because of the thinking he would kill me with that knife. I can talk again now so I will try to get home.

ADDITIONAL INCIDENTS OF HIS WANDERINGS

Shortly after he was returned to Pittsburgh a letter was received at his parents' home addressed to him from a Boston girl. The letter assumed acquaintanceship with him and asked for him to call at her home when the letter would be forwarded to him wherever he was. Writing to Boston I obtained, among other things, the following information:

My acquaintance with C. A. M. was much shorter than my letter would lead you to suppose. As a matter of fact, I met him only once and the letter was written on the impulse of a moment. I was returning to Boston from New York on the steamship *Canada,* Metropolitan Line. I was traveling alone and Mr. M. spoke to me about 9 o'clock and asked me if I minded if he talked to me as we both seemed to be alone. As I liked his appearance and was rather tired of having no one to talk to, I said, that I didn't object. The ship was to be convoyed part of the way home and as I was most anxious to see the destroyers we sat talking on deck until 11 o'clock. At that time the officers ordered all lights out, so we hastily said good night and went to our staterooms. During our conversation he told me that he was from Little Rock, Ark., and was a graduate of the University of Michigan, and that he had spent several years in Pittsburgh. He told me that he had been working in an airplane factory at Bayonne, N. J., testing the airplanes. He enrolled in New York or New Jersey, he didn't say which, for service in the Merchant Marine, Aviation Branch, and was on his way to Boston to enlist. He showed me the card sent him by the U. S. Shipping Board, requesting him to report at 128 Borden Street, East Boston. He asked me for directions to reach this address and that is how he happened to show me the card. He said that he expected to remain in Boston for two or three weeks and I intended to give him my address and invite him to call at my home, as he was a stranger in Boston. I am a Westerner myself, and as Boston people

Digitized by Google

are not the most hospitable in this country, I thought he might enjoy having one place to visit as he said that he knew no one in this city. I saw him but a moment or two the next morning, just as the ship docked. We just said, "good morning," and he told me that he had gone on deck at 1:30 to see the convoy ships.

This is the extent of my acquantance with Mr. M. I was sorry I had not given him my address as I thought him a very pleasant chap. Having a good memory I remembered the address at East Boston and called up the U. S. Shipping Board on the telephone to see if I could obtain his Boston address. They told me there was no aviation branch connected with the Merchant Marine. I told them that I was a friend of his and they gave me his Pittsburgh address, so I wrote to him there, expecting, of course, that the letter would be forwarded to him here in Boston.

Am very sorry to hear of Mr. M.'s trouble and if I have been of the least possible assistance by writing this letter, I do not mind the embarrassment it causes me, knowing that I have been unconventional in writing to a stranger and leaving others know it.

One other thing which I think I ought to mention. Mr. M. related a very peculiar incident with regard to a man named "Darol," I believe, as near as I can recall. He showed me this man's advertisement which contained his picture and a write-up about himself, together with a list of prominent clubs and colleges (Yale among them) where he has lectured. I think he calls himself a professor and claims to be a great psychic, scientific palmist, etc. This man lives in New York on Fifty-Second Street, I believe.

I do not care to write the details of the story Mr. M. told me about his acquaintance with this man but it sounded almost improbable. If it was true it might have something to do with his (Mr. M.'s) mental state. As the man is not normal, he may have done something to Mr. M. without his knowledge.

Have written all I know of Mr. M. which summed up does not amount to a great deal, but which may prove to be of some value. If Mr. M. should ever come to know of my part in the case, I should not care to have him know that I mentioned the incident in connection with "Darol."

If it would not be betraying professional confidence I would very much like to hear how Mr. M.'s case comes out. Did he see the letter I wrote to him?

In another letter this woman, to whom I sent the police picture taken in New Jersey, wrote:

Mr. Mason did not look to me as he does in the picture. The picture looks to me as if he had passed through a trying ordeal since I met him. Two or three things in his story (I sent her a copy of the diary) seem to relate to his trip

Digitized by Google

from New York to Boston. Where he speaks of the man who tore off pieces of paper, he may have referred to the fact that just before he spoke to me I was sitting by the rail with a magazine in my hands and was tearing off bits of paper and throwing them into the water. The enemy ships was probably our talk of submarines. He told me that he had no bag on board as it had miscarried. He was wearing the cap and a soft collar.

Inquiries made from the U. S. Shipping Board Offices elicited the information that C. A. M. had enlisted under his own name, had reported at Boston and had disappeared on the second day and was reported as a deserter.

The following additional information serves to round out our narrative:

The picture C. A. M. carried with him was one of his wife's taken three years before his disappearance. She had not put her name or address on the back of it. C. A. M., after he was returned home, on being asked concerning it, told us that it was the only thing "that" man (the man of the diary) returned to him of all his possessions and that he had written the name and address on the back; also that this man told him his name was M—; that he had all of his papers and his money when he was in the hospital; that he met this man after he left the hospital; that it was the Anna May Memorial Hospital, at Spring Lake, N. J.; that he was there two days and that he did not know why they did not notify his people if they had his papers. Both his father and his wife heard him make remarks in May about stock market losses but paid no attention to them, and that he was going to enlist if matters did not mend. His brother was along on the Greensburg trip but did not notice anything wrong. Before that, in April, however, he had what his mother-in-law calls a nervous collapse and for which he told her he had consulted a physician.

AN ANALYSIS OF PROBLEMS

Let us assume that C. A. M. suffered from a true amnesia and represents an actual dual personality. We cannot question that the duration of the struggle to evade the consequences of his acts and the intensity of his emotional reaction produced the kind of mental life which most often is responsible for a nontraumatic, nontoxic amnesia.

Digitized by Google

Though from the first he realized the nature of his acts, yet his deceit carried with it no heavy mental burden as long as it was fulfilling its ends. Yet some mental conflict was inevitable nevertheless. He was adjusting the daily situations but he could not thereby silence in himself the knowledge that some day this would be impossible. Whenever any one has an emotional inner life, which he cannot express to those around him, which he must keep to himself, and which is so intense that it influences all of his activities, it may in time become of such a degree as to lessen or suppress old associations, together with the power to form new ones. A memory seal is placed on the mind as far as its extenal projection is concerned. Yet memory is active, extremely active, in our hidden inner life. Subconsciously, it lives and functionates. It is in this way that we can conceive of the beginning of an amnesia. Intense mental suffering, if it exists and cannot get an outlet, in time must produce some reaction. With C. A. M. there daily grew the realization of ultimate exposure. Baffled desire is a potent enemy to a normal psychic life, especially when remorse steps in as an additional emotion. It is an emotion which deeply influences the self-regarding instinct of an individual. Inevitably, unless the circumstances are adjusted and the person can adapt himself to the situations as they actually exist, the problems or experiences or emotions which he attempts to bury carry with them other associated parts of his mental life and an amnesia results. The desire to forget, of course, in itself cannot produce more than a conscious forgetfulness of experiences. In normal life we see fragmentary glimpses of the dropping out of memories. Persons may dislike some one and thereby forget his name. We may have a disagreeable remembrance of a certain incident and submerge it out of consciousness. However, for an entire set of experiences to be blotted out, requires a more

Digitized by Google

powerful emotional basis than is found in normal life.

To illustrate: Mrs. M. went to the store one day leaving her 12 year old child reading. On her return she found the girl dying, her dress having caught on fire at the open grate. Mrs. M. after the death apparently did not grieve. She did not shed tears, or show the usual sign of mourning. After the funeral, I elicited from her that she did not accept the prevalent belief that the child was dead. It was some one else who had been burned and died. It was not her daughter who had been burned. This localized amnesia accompanied by the transference of the accident to some other person who did not exist, was manifestly different from normal forgetfulness. It could be looked on as a defense reaction, so that, when the recognition of the truth came to her gradually, she was safe from much mental suffering by being better prepared for the shock. Such a gap in self-consciousness without disturbing the woman's personality in any other way illustrates how deeply rooted feeling-states sometimes work. Amnesia is in no way different when it is produced in this way than is an inhibition of physical forces on a lower level which produces psychic anesthesia or paralysis. All of such phenomena are the result of a break in psychic associative continuity and are, therefore, called dissociations. We are unable to draw any conclusions concerning the degree of shock necessary, since the contents of minds differ, and their experiences vary. More than this, the cell stuff which makes up various brains varies greatly. The tenacity even of those primal trends which all of us have in common is a variable factor and has some bearing on the make-up of every person. The settings of the incidents in the life of every one are, however, much more important in determining his "behavior-patterns." The throwing out of moment-consciousness of a given occurrence is common-place and considered normal.

Digitized by Google

To be continuous, however, hardly requires more than to imagine the cause to be still working. If the life of a person is such that there seems to be no relief from its intolerableness and as a result there is lost the identification of self with the person who has these continuous emotions, the effects of which are being suppressed, some factor must be introduced into the situation to relieve the subconscious mind of its hurt in order to fuse the mental pictures and bring back a normal mental life. It does not seem to be always necessary for the intervention of a physician to relieve the situation except to a minor degree. Mrs. B.'s amnesia illustrates this:

THE AMNESIA OF MRS. B.

Mrs. B. was picked up by the police on the river bank, Jan. 1, 1919. She did not know her name nor where she lived. She thought, however, that she had a quarrel with her husband and that she went out to drown herself. The river looked too black so she sat by it. On an examination of Mrs. B., there were no physical evidences of any disease nor did we find any of of the so-called stigmata of hysteria. There was no evidence of alcoholism about her. In fact, she was very indignant at my pressing inquiries in regard to this. For nine days there was an absolute failure to identify herself. Newspaper advertisements elicited no response. No one applied at police headquarters asking them to trace a lost woman. January 9 I spent the afternoon with her and showed her how we dream actual events without realizing that they really occurred and that the fact that she thought she had quarreled and that she had tried to drown herself, revealed to me the fact that these incidents were true. If, I told her, she did not dream exactly who she was before the next day, I would have to put her to sleep and find out all about her former life in this way. I also told her to try to play a guessing game with herself after I left—to close her eyes and try to guess who she might be and where she might live, and memories would probably come back to her. That evening the resident physician of the hospital telephoned to me that she had called him and told him that she remembered her name and telephone number, which proved to be true.

The following incidents of her life, which I elicited from her in the next few days revealed the reasons for her amnesia:

Digitized by Google

She did all the housework for a family of fourteen, ten of whom were her children. Her family life was an unhappy one since the high cost of living had made it impossible for them to make both ends meet. She was unable to pay the routine house bills. Moreover, her husband, about a month before, got a piece of steel in his eyes at the mill where he worked and the little money he was receiving from the compensation board hardly served to feed the children. In addition to that she was worried by the physician's opinion that, perhaps, her husband would never regain his eyesight. The oldest daughter, who was married, was appealed to for help the day before she wandered from home but refused any assistance. They had quite a scene about it. A third factor in the few weeks before her disappearance was the presence of influenza in her home and her nursing at the same time four of her children day and night.

She has adjusted herself again to her existence. She feels now after what has happened to her that her daughter will appreciate and relieve her situation. Even if she does not, she tells me it is better for her to do her duty and she is going to try and not worry unduly about circumstances. You see how little I did in bringing about this readjustment.

CONCLUSIONS

It is obvious that C. A. M. had a partial dissociated personality which gave rise finally to a dominant one which was not his real self. It did not persist, however, and was easily fused with the elements of his actual self, so that a reintegrated C. A. M. resulted—and let us hope a better C. A. M. by reason of his experiences. A state of consciousness must always have memory and leave residual memories. That is, the brain mechanism conserves them and they are, as a rule, reproducible when the occasion requires. In amnesia they are merely not utilizable by the person at a given time; that is, they are submerged in consciousness. C. A. M.'s hidden emotional state produced both flight and concealment. Whether this concealment was shown by a partial volitional change of identity or whether his lost identity was a true dissociation, is really not debatable, since an opinion depends on the method of approach to the question. We believe, however, that C. A. M. did have an amnestic fugue which, for some days at least, altered his personality.

Digitized by Google

Whenever motives exist which force concealment of one's thoughts and anticipatory fear of disgrace is added to such a suppression, we have a state of mind which may effect a disintegration of it. Flight, change of address, of name and of occupation—do their presence prove adequate in themselves in explaining that a dissociation exists? Did he have fleeting moments of memory in which he wrote the address on the picture, gave his correct name to Miss D., etc., or were these intentional? Was his diary a fabricated story or the dramatic recital of a detached self? Were the Greensburg and other episodes before his disappearance the beginnings of a dissociation or were they preparatory, not only to his disappearance, but to a return home later on with his life-slate cleansed of all disgrace?

If one peruses the diary carefully, one finds much material for a justification of many of the accepted freudian mechanisms. There is distortion and dramatic condensation; detached dreamlike activities written down as actual occurrences; evidences of a mental conflict, as also of a desire to escape from intolerable situations; a desire, intense in its activity, for his wife revealing itself throughout the tale, and a compromise seemingly attempted at several points in the diary with the impossible situations. I need hardly discuss the places in the diary which point to these things. They indicate to me that, with C. A. M., there was something more than malingering, and that the above account represents a true amnestic fugue despite the acts which preceded it and which, standing alone, would indicate the possibility of his disappearance as being merely an attempt to escape punishment and despite his rapid return to normality while under my care at the hospital. One has no right to value objectively the motives and desires of an individual except at the actual time they occur. The roads C. A. M. could have taken after his acts led him to

Digitized by Google

disgrace were many. The law would see but one. Yet, as I have shown, the factors which would warrant malingering could and did produce a pyschopathologic state of mind. His earlier defense reactions and his flight may have been consciously effected, but his wish for disappearance of self was accomplished, for a short time at least, and a submerged self lived the scenes of the diary, as probably also those of Boston.

ABSTRACT OF DISCUSSION

Dr. Joseph Byrne, New York: I agree with Dr. Mayer as to the unsatisfactory status of psychotherapy. If scientific knowledge represents knowledge of a thing in its principle, then I must confess that so far the teachers and expounders of psychotherapy in this country have fallen down. If they had expressed clearly the principles of psychotherapy it would mean that almost any medical man could take hold of such principles and apply them. And yet we are lumbering along laboriously discussing the empirical therapy of individual cases in spite of the claim that psychotherapy has been established on a scientific basis. I think we and not the patients are the malingerers. What are we doing with our cases? An osteopath makes a positive diagnosis of a dislocated vertebra, fixes the patient on the table, pretends to punch a bone in place and so succeeds in persuading the patient that he will cure him and may actually cure him of the symptoms that immediately cause distress. We are struggling along, but after all what are we doing? Treating individual cases empirically without following clearly enunciated scientific principles, and then we find fault with the profession at large for their inability to keep psychoneurotics out of the hands of charlatans. If you listen to any man reading a paper on the mechanism of the psychoneuroses you will say he does not know what he is talking about. There is a way of distinguishing those malingerers, though I am not ready at this time to tell you what it is. I have a method that I will explain some day when I get around to it. The methods in vogue at present are confined to the personality of the individual using them. When a man gets up and reports a case in detail describing in full the wonderful results obtained but ignoring all reference to the basic therapeutic principles involved he is not a good scientist. What we want, however, is the expression of a universal rule which can be utilized by every practitioner in the detection of conscious or unconscious attempts at deception.

Dr. H. D. Singer, Kankakee, Ill.: In this discussion are we not going outside the realm of the science of medicine

Digitized by Google

altogether? When the question is raised as to whether certain manifestations are psychoneurotic or malingering what is it that we are trying to establish? To me this means nothing more than the question as to whether the individual selected this particular mode of meeting some situation with consciousness as to its nature and consequences. In other words, the problem is one purely of ethics or responsibility which is not medical. To us, as physicians, the essential fact is that the patient has shown a certain kind of reaction under certain conditions and we are not concerned with the question of responsibility at all. Even the patient with hysterical amnesia can be forced, by appropriate measures, to recall the incidents apparently forgotten and hence must have been conscious at the time of their occurrence.

Dr. W. A. Jones, Minneapolis: There is another phase, or factor, in the causation of amnesia, that has not been mentioned. The majority of cases, however, are conceded to be of mental origin, due to physical disorders or structural changes in the cortex and pathways. A man, 55 years of age, had been a steady drinker for many years, and he was not very careful as to his way of living. He finally developed a transitory amnesia. He forgot the names of his employees and associates. He occupied his time at his desk in drawing figures, or words, waiting for his attack to pass off, which it usually did in two or three hours. He was finally put through a searching examination, and was declared free from organic disease by the internist, but was permitted to continue his drinking. After this, he consulted another physician, who cut his liquor off entirely, and put him on a simple diet, clearing out his intestinal tract with repeated doses of calomel. The result was a clearing away of the amnesic attacks. There are some cases of amnesia that are due to somatic causes.

Dr. Albert E. Sterne, Indianapolis: If we analyze these cases properly, we will find that to many it is a way out of an intolerable situation. The war situation at the front and even behind the front and on home soil, was intolerable to thousands. These cases show an exaggeration of the tendency which we all habitually manifest, to side-step a disagreeable situation. There are cases, however, where the amnesia is a true one, where there is no motive that can be discerned by the most careful questioning. We have cases of somnambulistic automatism in which there is a sharply defined loss of consciousness, in which the automatic phenomena go on, where there is only conscious registration taking place, which in consciousness the individual cannot recall. We have all seen such cases. I have noted the transition into the amnesic state and again out of the amnesic state for which no motive could be elicited. These conditions have a close resemblance to that form of lack of consciousness which is called "psychic epilepsy." Many of these cases are of serious

Digitized by Google

portent. I recently had under observation a young man, an officer who did not get to the front. He was in the Spanish-American war and after that joined the British army, went through the Boer war and while in Africa acquired "black-water fever." He was sent to Europe from Africa by the British medical authority and recovered his health. He enlisted in the American army and was sent to Camp Taylor. On Christmas Eve, 1917, he started to visit his wife, fell on the ice and struck his head, but was not seriously injured apparently. After spending the evening with his wife he came back to camp and on the way completely lost himself. He was picked up by the M. P. at the camp, but could not tell who he was or what his division was. He was sent to the hospital for about three months. He finally recovered and was discharged as unfit; spent several months at his home, reenlisted and was sent to another camp. While in Chicago he had no desire to escape service. He was finally discharged and came back to Indianapolis, resumed his practice of law and, while on the street, suddenly lost himself again. This period lasted about one hour. He was referred to me and I examined him thoroughly. He showed a syndrome of organic cerebral lesion. The first blood serum Wassermann was positive. Spinal puncture showed extreme pressure, slightly increased cell count and negative Wassermann. A second blood test taken at the time of spinal puncture gave a negative Wassermann. Examination by three different experienced serologists gave two negative and one positive Wassermann of the blood, taken at the same time. He has refused intensive treatment. The question is, is this an activation of a dormant syphitis through traumatism, which account for these vascular attacks or is this man developing an epilepsy? He denies syphilis. Could it be possible that the protozon infection of twenty years ago, "black water fever," may have been yet in play at the time the serum was positive? I do not believe we should wave these cases lightly aside as evidence of malign motives, or that we should look on them all as cases of hysteria. Many of them are potential epileptics who are going to come to very serious grief later on, or activated syphilitics who require careful treatment.

Dr. Tom A. Williams, Washington, D. C.: With reference to the interpretation of these amnesias, we must recognize the fact that many hysterias have taken their origin from suggestion which has arisen from physical conditions. Thus in the cerebral commotion created by violent explosion the first phenomenon is loss of consciousness. As this is recovered from a state of obnubilation supervenes in which there is marked loss of memory, not only for recent events, but in some cases preponderantly for early events while the events of the war may be recollected. In others, isolated portions of experience only may be forgotten. The picture

Digitized by Google

is infinitely variable and contradicts the law of Ribot about the regression of memories being inversely proportional to the duration. Just as is the case in individuals reported in my studies on traumatic and occupational neuroses where body conditions provided the suggestion for the hysterical manifestations even after the cure of the body condition, so in these commotioned patients they may continue to believe they have lost their memory even after the physical disturbance has disappeared. The man has not enough dynamic volition to compel himself to the effort required to recollect once more. It is in cases of this kind that intensive persuasion causes a response in the memory of events which the patient believes still forgotten, and which really have been forgotten at first As they begin to recover, they recover their memory when reeducative persuasion is applied. In some cases a powerful emotion is capable of wiping out a memory momentarily. The effect on the patient's mind, however, may be so great that he will not spontaneously make the effort to recall what has been forgotten and remain in the belief that he has lost his memory just as is the case in others after a physical injury. Credulous acquiescence in the statements of these patients leads physicians to elaborate the simple picture presented by the patient, whereas a proper skepticism and an intensive reconstruction of the souvenirs leads to rapid cure.

Dr. Edward E. Mayer, Pittsburgh: Dr. Byrnes in his criticism of physicians speaks of an idealistic condition which is not realizable. An inductive approach to our problems is proper but at present we must utilize ideas whenever our facts do not answer. In respect to Dr. Singer's remarks, I agree with him that the question of responsibility should not be entertained from our point of view whenever we consider a psychopathologic problem but we are compelled to by our legal friends. For this reason I brought up the question of the validity of our present day conceptions in reference to true or false amnesia and how we can distinguish them independently of any bias.

Digitized by Google

SOME EXPERIENCES IN NEUROLOGIC THERAPEUTICS

CHARLES K. MILLS, M.D., LL.D.
Emeritus Professor of Neurology, University of Pennsylvania School of Medicine; Senior Neurologist, Philadelphia General Hospital
PHILADELPHIA

PERKINISM AND BURQUISM

Toward the end of the eighteenth century a Connecticut physician, named Elisha Perkins, aroused much interest among the medical profession and the public, especially the latter, by a pamphlet in which he advocated the use of what were termed "metallic tractors" in the treatment of a variety of diseases. The tractors were small rods of different metals the extremities of which were applied to parts supposed to be diseased. Wonderful cures were reported and exhibitions of the value of the tractors were given throughout the country. The fame of the treatment crossed the ocean to Denmark, England, France, and other parts of Europe. Books and pamphlets concerning it were published, but after a decade or two it passed into innocuous desuetude.

Many years after Perkins and his disciples, at first at Milan and afterward at Paris and elsewhere, one Dr. Burq appeared and advocated the use of metals both in the diagnosis and in the treatment of neurotic affections, his efforts receiving attention under the designations of metalloscopy and metallotherapy. Burq published a small book in 1882. He was taken up by Charcot after he had been extensively patronized by the public.

In the ninth decade of the last century I became much interested in the publications of Charcot and Richer on the subject of hypnotism and also in Bur-

Digitized by Google

quism, which belonged after all in the same domain. I had a series of metallic disks of uniform size made—of copper, zinc, tin, iron, lead, silver and other metals. These are probably still to be found stowed away in some closet at the Philadelphia General Hospital. For my experiments with gold I generally used a twenty dollar gold piece, which, I presume it is unnecessary for me to explain, was not left at the hospital.

Experimentation was carried on not only as regards the external metalloscopy and metallotherapy of Burq, but also into what was termed internal metallotherapy. Disks first of one metal and then another and another were applied to blindfolded patients, especially hysterics, and when the metal to which the patient responded—that is, which created some peculiar sensation under the point of application—was found, this metal was used to dispel, transfer, or in some instances to produce anesthesia and other phenomena. The method was used in efforts to relieve contractures as well as anesthesias and paralyses of presumably functional type. Sometimes when I found that the patient was responsive to a particular metal, I would administer in addition to the external applications, this metal or some of its salts internally (internal metallotherapy). The results, as might be expected, were somewhat irregular, although at times they were rather striking. Soon, however, I tired of these performances, as of my various experiments in hypnosis, which were often interesting but rarely resulted in any permanent good to the patient. One thing they did was to strengthen the views that I held, even then, as to the importance of suggestion and countersuggestion.

THE SUSPENSION TREATMENT OF LOCOMOTOR ATAXIA

Motchoukowski of Odessa published a pamphlet in 1883, describing the advantages of using suspension in the treatment of locomotor ataxia, having arrived at his ideas through the accident of observing a patient

Digitized by Google

suffering from this disease apparently obtain relief from the application of a plaster-of-Paris jacket. Professor Raymond, traveling in Russisa, observed Motchoukowski's results and brought an account of them to Paris.

Charcot, whose endorsement brought Burquism into more or less undeserved repute, was also responsible in considerable part for the attention which the neurologic profession gave to the treatment of tabes by suspension. He and his assistant, Gilles de la Tourette, experimented with Salpêtrière patients with what were believed to be gratifying results. The improvements reported by Motchoukowski and Charcot in some of the ataxic patients were said to be remarkable. Some of these were changes for the better in the Romberg symptom; improvement of gait in walking; diminution of lancinating pains; beneficial effects on sexual power; and better control of the bladder. As might be expected the absent knee jerks and pupillary phenomena were not favorably affected.

Neurasthenia, Friedreich's ataxia, and paralysis agitans were other diseases reported as improved by suspension. The rationale of the improvement was not explained with any clearness. It was supposed, however, that the spinal circulation was benefited and that some stretching of nerve fibers occurred, the latter supposition being altogether doubtful. At that time the fact was not generally recognized, as at present, that locomotor ataxia, like general paresis, is a true syphilitic disease due to the presence of spirochetes.

The apparatus used by Charcot was that contrived by Dr. Sayre of New York in the treatment of his cases of spinal caries by suspension. Charcot first published his results in a lecture given Jan. 15, 1889. It is noteworthy that many patients with tabes reported as having undergone this treatment expressed a sense of having been much benefited by the suspension. This is probably to be explained by the well known optimism of tabetics.

Digitized by Google

Dr. S. Weir Mitchell, in the *Medical Times,* April 13, 1889, described an improved apparatus for suspension, the chief peculiarity of which was that the suspension was in part made from the elbows, the arms being held to the side by a strap, and additional suspending apparatus being applied to the chin and occiput.

Cases were reported to Philadelphia societies as to many others. The Orthopedic Hospital and Infirmary for Nervous Diseases, of this city, and not a few other institutions, were supplied with suspension apparatus, and some enterprising neurologists saw to it that their offices were similarly equipped. The merry dance—or rather the merry hanging—went on for a time with what was supposed to be advantage to the suffering tabetic. This treatment soon passed to the therapeutic limbo.

THE SURGEON AND EPILEPSY

With epilepsy I have had many interesting experiences, both medical and surgical. I will speak here only of the latter. From times remote, considered in the light of a century or a half century, numerous suggestions and experiments have been made regarding the relief and cure of epilepsy. Very early in my professional life I knew something of the efforts that were made on the basis of the old "reflex" hypothesis to rescue the unfortunate epileptic. Children were circumcised; women were deprived of their ovaries and occasionally of the clitoris; a nerve in the hand or the foot or at the root of a tooth or somewhere else, according to the apparent indication, was resected, and much more of a similar sort was done to exorcise a constitutional disorder which refused to respond to the rites employed to banish it.

The term epilepsy is here used in a broad sense to cover several forms of convulsive seizures. Long ago the trephine or trepan was sometimes used without any particular care or skill or selection of site for the relief

Digitized by Google

of spasms. Bloodletting was at one time so heroically employed in the hope of relieving epilepsy as to become almost entitled to be included under surgical procedures.

When, some fifty years ago, the modern doctrine of cerebral localization obtained a sure footing in physiology and neurology, operations soon began to be suggested and performed, the surgeons being guided as to the choice of site by the principles of localization. Cranial fracture, localized hemorrhage, abscess, and tumor became the frequent objects of surgical intervention, often with marked or even brilliant success. It is not my intention, however, to go into this subject at any length. I merely refer to it to introduce a few remarks regarding two or three of the surgical procedures which since then have been generally laid aside.

One of these was cortical excision, which was advocated by Horsley and others, and for which I was sometimes responsible. The idea in these cases was that even in the absence of any clear evidence as to a gross lesion, if the spasm could be shown to be of a local or jacksonian type, trephining should be employed. After fixing the exact center or area likely to cause such spasm by means of faradization of the exposed cortex, this center or area was then excised. On the whole, the operations of cortical excision, of which a considerable number were performed, did not prove successful, although in some instances the spasm did not recur for a long time after the operation. One of the results in these cases was, of course, that a local paralysis in the limbs or part controlled by the centers excised necessarily occurred. This in most cases became less and less marked and would not have contraindicated operation if a positive relief of the spasmodic attacks had been obtained.

I had brought under my observation a number of cases of so-called idiopathic epilepsy in which operation was suggested or in which an opinion was sought

Digitized by Google

as to the propriety of such operation. In these cases jacksonian spasm; that is, a monospasm or hemispasm involving the face or arm or leg, or one or two of these parts, was usually present as part of the general motor paroxysm. I soon learned that a frequent, if not the most frequent, source of so-called jacksonian spasm was idiopathic epilepsy, a fact to which attention was called by Collier. As I spoke of the matter in one of my papers, the jacksonian attacks occur inside the general epileptic seizure and their presence simply emphasizes an opinion long since expressed by that great neurologist, Hughlings Jackson, that if epileptic fits were sufficiently well observed it would be found that in nearly all cases such seizures began with some form of local spasm. This, of course, is often overlooked and even acute, sharp-eyed observers, whom I have frequently employed in my wards, have sometimes failed to note its occurrence, so quickly does the initial spasm radiate to other parts and eventually to all portions of the body musculature. In a number of cases which came under my observation and in which I was responsible for the procedure, trephining, guided by these jacksonian manifestations, was used, no lesion being revealed. In some cases, however, considerable benefit in the way of suspension of the paroxysms occurred.

In recent years the surgeon has extended his efforts for the relief of epilepsy to the abdominal cavity. One distinguished surgeon, some dozen years ago, recommended for the relief of epilepsy the operation known as appendicostomy. Various considerations were advanced in the discussion of epilepsy at the time of this recommendation, but into these I cannot take the time to go. They were based chiefly on the idea that epilepsy was due to some form of intestinal toxemia. A hole was made into the intestine in the appendical region, and a course of irrigation of the colon was begun, usually with warm water. This, of course, was at first under the direction of the surgeon himself and

Digitized by Google

of the nurse, but later, in some of the cases in which this treatment was employed, the patient was taught to treat himself while lying in bed, by passing a tube through the opening into the intestine and then allowing slow irrigation to occur, the fluid and whatever it carried with it being received in a bed pan or other receptacle. A very few striking results were reported; that is, cases in which the epileptic or epileptiform attacks had ceased for several months or perhaps longer. So far as I know, no results of absolutely permanent value were obtained. This was a rather striking procedure, but one, it always seemed to me, not likely to become acceptable to the patient who was expected to keep on with this process of irrigation on his own hook.

A western surgeon more recently described the operation of cecostomy which he used in cases of constipation mechanically produced, and also for the treatment of epilepsy. It was practically the same operation as that to which I have just referred, except that the opening was perhaps an inch or two away from the appendical orifice.

The same skilful and distinguished surgeon, in the course of his operative procedures for the relief of constipation mechanically produced, came across some constipated epileptics in whom the frequency of the attacks was much diminished by the operations performed, which were usually on the colon and need not here be described.

Going a little further into the subject of epilepsy, he learned, what I am sure has been the common knowledge of neurologists, that most epileptics were subject to constipation and therefore, perhaps, the constipation and the epilepsy had the relation of cause and effect. He advocated procedures similar to those which were used for constipation in nonepileptic patients. Noting that, while epileptics were generally of constipated habits, the multitude of the constipated were only in

Digitized by Google

rare instances the victims of epilepsy, he found it necessary to look a little further into the subject. Doing this by methods which are well known to the profession, he came to the conclusion that epilepsy was due to some form of bacterium or bacillus which probably originated in the duodenum, and whose habitat eventually became the great intestine, or colon. The steps that followed were rather extraordinary and yet I suppose logical from the standpoint of this observer. Not being able always to establish a proper fecal current through the torpid, diseased and absorbing colon, he came, in the process of time, to the idea of getting rid of the colon, and in this way the operation of colectomy was introduced, and not a few epileptics were reported as cured or greatly benefited by this extirpating operation.

Personally I cannot help thinking that this large part of our intestinal tract called the colon has some valuable function in the average human being, and while it may be a convenience to the individual at times to have his bowels short-circuited from the ileocecal region to the anus, this procedure may have its physiologic and pathologic drawbacks.

Eventually the surgeon to whom I have referred, because of the negative results of laboratory investigations made by others, and also under his personal supervision, was obliged to withdraw his endorsement of the idea that the special convulsion-producing germ had been found in the intestine.

DENTAL INFECTION AND THE PSYCHOSES AND NEUROSES

Some years ago I presented a brief paper before the Stomatological Society of Philadelphia on "Obsessions Regarding the Mouth and Teeth." After discussing the subject of obsessions in general I gave details of a few interesting dental cases. One patient had the idea that she was suffering from some discomforting affec-

Digitized by Google

tion at the root of a tooth. This was removed. The distress then began to change its dental habitat, and the patient went through a series of tooth-removing campaigns, but still no relief was obtained. When I first saw her she had only one tooth left, and this was soon extracted by a dental surgeon. At last a distinguished oral surgeon engaged in a vain search for her elusive pain by probing the antrum. She became an alcoholic habitué, and eventually died broken down by her obsessions and her excesses.

In another case the mind of the patient was turned to her teeth until they were all removed and plates were substituted, and later, from time to time, no relief being obtained, she had one plate after another made until the number had increased to twenty-two.

It has seemed to me in recent years that the obsessions regarding the teeth have taken possession of the medical profession. Numerous papers have been written about the rôle of focal infection originating at the roots of the teeth in the causation of mental and nervous maladies. Teeth by the score, many of them of excellent quality, have been sacrificed, and in not a few instances cures or great benefit have been alleged as a result of the treatment. The matter is one of much importance, and has been emphasized by my observation of a number of victims of the dental forceps.

Dementia praecox, manic-depressive insanity, epilepsy, neurasthenia, hysteria, and psychasthenia are only a few of the diseases that have been attributed to dental infection.

The usual procedure in private practice is for the patient, who has probably been seen by one or several neurologists or alienists, to seek, because of the wide exploitation of the subject, a physician who is known to lay stress on the importance of tooth infection. The patient is then sent to the roentgenologist, who almost invariably discovers and is able to mark out a series of alleged abscesses, which are in some instances at least,

Digitized by Google

absorptive lacunae, innocuous foci, or mistaken observations. I do not mean to say that real abscesses are never demonstrated, but as one of my dental friends has pointed out to me, these are frequently not to be found even by the probe in the hands of a skilful surgeon. In a majority of cases, however, the teeth are sacrificed on this altar of focal infection.

The tooth extraction sometimes has an unintended outcome. The patient's mind, already obsessional from the very nature of the malady, becomes fixed on the teeth as a source of his ailments, and what is perhaps worse, the same obsession takes hold of the minds of relatives or guardians. After three apparently good teeth had been removed from one of my patients, she, like the first patient cited in this section, became possessed with the idea that the work of tooth extraction should go on, this obsession pursuing her at all times.

In another case of dementia praecox, as I was told by the mother and the caretaker, the boy had his teeth roentgenographed. Nothing was found, but the advice was given that it would be a good thing to remove a couple of teeth anyhow.

One of my medical friends had a rather interesting experience in connection with this subject. An old gentleman, who was not only a patient but a family connection, had suffered with pain in the neighborhood of the sacro-iliac synchondrosis, and had been treated therefor by the usual therapeutic procedures for affections of this locality, with, however, only partial relief. A consultation came about with a physician and a roentgen-ray specialist, and one of the consultants without any examination of the mouth or jaws expressed the opinion that the probable source of the trouble would be found in abscesses at the root of the patient's teeth. My friend, after allowing him to go on with his dissertation a short time, said casually, "It may be, but I must remark that our patient hasn't had a tooth in his head for the last five years."

Digitized by Google

LINEAR CRANIECTOMY AND OTHER METHODS OF CRANIECTOMY FOR THE RELIEF OF IDIOCY OR IMBECILITY

Lannelongue of Paris[1] reported his experience in two cases of microcephalic idiocy in which he performed the operation of linear craniectomy. One of the patients was apparently benefited, although, as the recorder notes, training of the patient was resorted to in addition to the surgical procedure. My first experience with this operation was with Dr. W. W. Keen of Philadelphia, who, shortly after the publication of Lannelongue's article, began to make use of the operation for cases similar to those described by the French surgeon. The usual procedure was, with certain technical precautions, after lifting a flap of the scalp of sufficient length and breadth, to cut out a strip of bone about one-fourth inch in width and about 4 to 6 inches in length. Dr. Keen published several articles describing his experiences with this operation. As he has recently informed me, he finally gave it up, the results obtained not justifying a continuance of the surgical measure.

Dr. C. L. Dana of New York published two articles on the subject.[2] He had the surgeon undertake the procedure described by Lannelongue and Keen with, however, some modifications as regards the character and extensions of the bony removals. Dana, like Lannelongue and Keen, believed that he obtained a few good results which strangely enough he ascribed to the "pedagogic" influence of the surgical procedure. I must confess that this explanation never strongly appealed to me. It seemed to savor of the explanation of the treatment of delusions by the repeated administration of shower baths until the insane subject finally concluded it was better (for the time being, at least) to give up his delusions rather than further endure

1. Lannelongue: L'Union méd. Series 3, **50**: 42 (July 8) 1890.
2. Dana, C. L.: A. J. M. Sc., New Series **110**: 24 (Jan.) 1896.

Digitized by Google

the punitive measures. These delusions usually promptly returned when the patient was assured he was out of further danger from the cold douching. The operations were also to some extent reminders of the disciplinary measures—carried out with clubs, whips, or even incisions—in order to improve the manners and morals and presumably the intellects of obdurate criminals.

The wonderful theory, however, advanced to support this procedure was usually not pedagogic or disciplinary, but was based on the idea that by removing strips or variously shaped pieces of bone from the cranium the retarded brain of the microcephalic or demimicrocephalic idiot or imbecile would then be given an opportunity to expand and develop. It was sometimes argued that the fontanels and sutures of the infantile skull had closed prematurely, thereby holding in check a brain that would otherwise develop to fair proportions. The argument in favor of the operation, however, was, to my mind, founded entirely on false premises. It entirely overlooked the fact that the arrest of the brain, like the arrest of the skulls in these cases, is due to biopathologic processes, hereditary and embryonal. As my colleague, Dr. James Hendrie Lloyd, once remarked during a discussion of Lannelongue's operation at the Philadelphia Neurological Society: "It is not possible to improve the quality of a bad nut by making a hole in the shell which encases it."

My chief object for recalling this operation, as in referring to much else in this paper, is to show how prone neurologists and surgeons have been to have recourse, without due consideration of principles, to procedures that could not possibly be of any practical service.

Operations in porencephalic and hydrocephalic cases have proved as futile, in my experience, as the craniotomies for the microcephalic. It proved a great

Digitized by Google

temptation to myself, as well as to others, in the earlier days of my localization experiences, to operate in cases of convulsion, with or without mental deficiencies, when the sites for such operations could with accuracy be easily indicated. In the main, these cases were examples of porencephaly, the residua of previous hemorrhages, or of focal encephalitis after various infective disorders. Due consideration was not given to the significance of the cavities, which were in fact in most instances, whatever their origin, conservative or partly protective. Many of the cases ended disastrously, if it can be regarded as disastrous to have such patients perish in the hands of the neurologist and surgeon. In a limited number of cases in which the cysts were shallow some good seemed to result, but in others, if the patients escaped death, there was often an increase in the excitability of the cortex.

Tapping the ventricles for hydrocephalus was in my experience equally lacking in efficacy, even when the operation was accompanied, as it infrequently was, by an imperfect effort at slow compression of the skull.

NERVE STRETCHING

About thirty-five years ago, the operation of nerve stretching for a variety of sensory and motor disorders began to be used. John Marshall in 1883 delivered the Bradshaw lecture before the Royal College of Surgeons of England on "Neurectasy or Nerve Stretching for the Relief or Cure of Pain." This lecture, which was carefully prepared and founded in considerable part on experimentation, helped to give the operation a scientific status. Dr. Christian Fenger[3] of Chicago and Dr. W. W. Keen of Philadelphia, among others, made valuable contributions to the subject of nerve stretching. Unquestionably this operation was sometimes successful in relieving severe pain, especially when it seemed of a purely neuralgic

3. Fenger, Christian: Collected Works of Christian Fenger, M.D., 1840-1902, Philadelphia, W. B. Saunders Company, 1912, Vol. I.

Digitized by Google

type. The operation was also resorted to occasionally, but with less favorable results, for tabetic pains.

Among the motor disorders in which nerve stretching had varying success, were facial tic and spasmodic torticollis, many cases of the latter affection really belonging in the category of tics, although this was not at first generally recognized. I recall two cases of facial tic in which operation with good results was performed by Dr. Keen. The stretching force exerted in these cases usually amounted to 4 or 5 pounds of weight. The nerve was exposed near the stylomastoid foramen, and the stretching was done steadily—not with sudden force. The first result of this operation on the facial nerve was complete paralysis, the tic disappearing. The paralysis was gradually recovered from, and in one or two instances which fell under my observation, without return of the spasm. This, however, was not the usual result, and the operation was soon largely abandoned. The cause of such tics was either partial nuclear degeneration or a faulty cortical discharge, neither of which could be banished by operative procedure.

I had considerable experience with spasmodic torticollis. One of my earliest papers was concerned with this affection. It is not necessary to tell an audience of neurologists that this is one of the most intractable of diseases. Stretching of the spinal accessory nerve was employed for its relief, but without success. Equal want of success, however, attended other operations, as for instance tying the accessorious with silver wire, sectioning it, cutting the muscles presumably involved, and excising the spinal nerve roots. The lack of success in this operation seemed to be due to the fact that the spasmodic disorder was one which probably had its seat in instability of the motor cortex. When the affection was closely studied it was found to be not a neural or muscular affection, but a disorder of movement involving a group of cooperating muscles.

Digitized by Google

Shortly after the period in which nerve stretching was advised and resorted to for the relief of pain and local spasms, the operation began to be recommended in the treatment of several forms of sclerosis, as symmetrical lateral sclerosis (a rare affection), disseminated sclerosis, and spasmodic paraplegia. In one of my cases of disseminated sclerosis nerve stretching in the lower extremities by the method without cutting; that is, by forcibly flexing the extended leg on the trunk, was used, and no beneficial result having been obtained, Dr. Keen stretched the sciatic nerve by a cutting operation in which after exposing the nerves he subjected them to a slowly continued pull corresponding to about 25 pounds of weight. This operation, as might have been expected, also resulted in failure.

Looking backward on these surgical procedures, their futility is easily recognized. It is evident that, at the most, they could have only temporarily affected the spasmodic disturbance. In other words, the operation was not based on a proper consideration of the nature of the disease, and therefore was not a really rational procedure.

GLANDULAR THERAPY AND ABDERHALDENISM

Foremost among the methods of glandular therapy to be tried out by me personally was the use of preparations of thyroid. In a well known line of cases these proved invaluable, as in myxedema and myxedemoid and cretinoid cases, in adiposis dolorosa, and in forms of adiposis not of the Dercum type. This treatment in a strict sense was not curative, but when the amount which could be administered without too much constitutional reaction was determined by trial, the various preparations of the glands of the sheep or of other animals were given with continued advantage and sometimes with remarkable success. As a rule, however, it was necessary to keep up the administration as a part of the regular diet of the patient.

Digitized by Google

In a limited number of cases of epilepsy I have had decidedly beneficial results from the use of thyroid with bromids and arsenic and special forms of diet, as for instance the so-called purin-free diet. In addition to thyroid, pituitary preparations have also proved of value in cases of epilepsy with dyspituitarism.

With preparations of other glands, like testicle, ovary, mammary gland, and suprarenals, my experience has not been promising, although I have seen some benefit accrue from the use of suprarenal or pancreatic extracts, especially in melancholia and epilepsy.

In connection with this question of glandular therapy, that of glandular removal comes naturally into the foreground. Every neurologist and alienist will remember the rage in the early days of safe, that is, of aseptic and antiseptic, surgery for removing ovaries for the relief or cure of neuroses and psychoses. The fashion was so rampant for a time that there promised to be partial race suicide by way of the surgeon's knife. Directly or indirectly, I came in contact with much of this work. My experience might be included in the simple statement that nine tenths of such removals did not result in the relief of nervous maladies. In a considerable percentage of the cases the postoperative mental or nervous state was such as clearly to indicate that the operations had done harm rather than good. These operative procedures in some instances were the source of traumatic affections, especially troublesome adhesions, and in other cases of new or more confirmed obsessions. It is rather interesting to note the contrary manner in which the two sexes were treated as regards the sexual glands. The woman suffering from a variety of mental or nervous disorders had her ovaries removed, while, forsooth, the man afflicted in the same or a similar manner was treated by the administration of testicular preparations. So far as results were concerned, how-

Digitized by Google

ever, it mattered little; one was about as effective as the other.

When a few years ago (about 1912) the investigations and theories of Abderhalden of Halle were published, a new stimulus was given to glandular therapy. I do not feel competent to pass fully on the subject of Abderhaldenism, as I have found it somewhat difficult in the limited time and opportunity at my disposal to determine from scientific considerations the value of the methods both of diagnosis and of therapeutics suggested by a study of the protective or defensive ferments of the body. Some of my own patients, and other patients of whom I have knowledge, have been subjected to these investigations and have had made on them therapeutic applications of the results of such investigations. The cases to which I refer particularly include examples of dementia praecox, melancholia, and hysteroneurasthenia, especially the first.

As the result of painstaking Abderhalden tests, guarded by all the precautions suggested to those who work in this field, certain glands have been determined as most likely to be beneficial because of their supposed or known antagonistic action to other glands, which the investigator has found to be dysfunctioning. Preparations of testicle, for instance, have been used when the pituitary was the offender; of the suprarenal when the suprarenal was at fault; or of the pancreas when the indications were of pancreatic infantilism or deficiency. For instance, certain glands are given to replace a supposed deficiency in the corresponding glands, and their opponents are administered to subdue hypersecretion. Results apparently beneficial but usually temporary were obtained in some of these cases. In a very few instances more permanent results were in evidence. I have never been thoroughly convinced, however, that the clinical course of a genuine case of dementia praecox was halted for more than a brief period—a period similar to that which one

Digitized by Google

sometimes observes in these cases without the use of Abderhalden or any other form of therapeutics.

The observations of Ludlum on psychopathic patients cured or greatly benefited by the use of either acid or alkaline preparations—according to the result of investigations into the acidity or alkalinity of the saliva, urine, stools, perspiration and blood—in combination with preparations of ovarian gland when the blood pressure is high, and of suprarenal extracts when it is low, are of decided interest and open up a field of therapy for the neurologist and alienist.

One of the points which has always impressed me when studying reports of this sort and the theories on which the treatments suggested in such records have been based, is that due consideration is not given the teratologic origin of the affections which it is hoped to cure by changed acidity or alkalinity, or by these combined with the use of glandular preparations. The fact is too often overlooked that the glandular condition, and even the state of acidity and alkalinity in some instances, are themselves only a part of an ordained embryonal state which cannot be successfully combated, or at least cannot be attacked with full success on chemical and other theories of such limited import.

1909 Chestnut Street.

Digitized by Google

THE RANGE OF THE GENERAL PRACTITIONER IN PSYCHIATRIC DIAGNOSIS

E. E. SOUTHARD, M.D.
BOSTON

One cannot help marveling at the progress made by psychiatry in the American medical scene. But the standing of psychiatrists and the prominence of psychiatric topics among medical men at large is more than paralleled perhaps by the vogue of psychiatrists and psychiatry in the broader circles of mental hygiene. It has been my privilege, as delegate from the Commonwealth of Massachusetts, to attend in the last few years many meetings of nonmedical workers, such as meetings of social workers, psychologists, general scientific workers, and even of teachers of philosophy; and I can report that this associate medical world (if we may call it so) is ready to go more than half way in meeting physicians on a mental hygiene platform. Too eager to enter on unsuitable tasks are these nonmedical workers, some advocates of a Little Medicine might aver. But the Greater Medicine should open its arms to what Weir Mitchell called the "assistive" arts and sciences and, in matters touching the community, give over all that savors of a closed shop for M.D.'s only.

Though the face which psychiatry presents to the "assistive" nonmedical branches of mental hygiene is most respectable (the osmotic interchange perfect, one might say), the interior relations of psychiatry to the rest of medicine form something of a problem. One difficulty, which I do not here more than mention,

Digitized by Google

is the proper adjustment of the planes of contact between clinical neurology and psychiatry. The research basis of these two divisions of practical medicine forms a continuous unit, having structural and functional aspects, to be sure, but nevertheless growing steadily together.

Leaving the interrelations of neurology and psychiatry to themselves for the moment, I come to my chosen topic. I consider that, all in all, the appreciation of psychiatry by the general practitioner has not kept pace with the increasing appreciation of it now being shown by neurologists and with the even greater advance in the esteem in which psychiatry is held by social workers, psychologists, philosophical and ethical teachers, and other practical and scientific types of nonmedical mental hygienists. In short, the general practitioner ought to enter consciously and confidently the field of mental hygiene, some of the tasks of which lie very near his hand.

But why is neuropsychiatry caviar to the general practitioner of medicine? It is also, we must admit, caviar to the general theorist, in American medicine at least. A determined effort to overcome the stepmotherliness of practitioners and theorists toward the general topics of this section of the American Medical Association must soon be made, if we are not to be quite outstripped by other nations even on the barest practical lines. I do not assign our unpopularity to inborn deficiencies of American practitioners. I am not even sure that our medical theorists are altogether responsible for averting their eyes. I am a little more inclined to fix the blame on the pedagogic group in our medical schools. Not that appropriate curriculums fail to be arranged, and not that clinical opportunities fail to be thrown open to medical students. But there is one the part of teachers in other branches than the neuropsychiatric an almost constant avowal of their lack of interest in nervous and mental diseases. This avowed apathy can hardly miss exerting its effect on

Digitized by Google

the weary medical student, looking (as is his duty) for *things not to learn*. But what is the cause of the pedagogic look askance at nervous and mental diseases? Some of our own specialistic colleagues, who talk glibly of everything as a defense reaction, intimate darkly that neuropsychiatry is a sore subject with most medical teachers because they are so ignorant thereof. Be that as it may, I feel that not all the blame attaches to our nonpsychiatric colleagues, and that our own obscurantism has blocked progress.

How much can be done with a frontal attack is doubtful. But correspondence following a paper presented to this section last year on a similar topic led me to think that a flank attack on medical teachers and research controllers might not be necessary to secure greater interest in mental hygiene on the part of the general practitioner. In the first place, the psychiatrist is, in a different way, almost as general in his approach as the general practitioner. The map quality and representativeness in the nervous system of virtually all other systems in the body insures a consideration of many things by the psychiatrist after the precise manner in which a general practitioner faces the same situation. This generality of view (e. g. that a psychiatrist, like a general practitioner, has to be something of an endocrinologist, a good deal of a syphilographer, etc.) is just as advantageous and just as dangerous as the generality of view which it is the duty of the general practitioner to have. Again, the psychiatrist, far more than any other specialist (unless perhaps the obstetrician), has to take his patient as a total person, as a globar individual—and this is precisely the duty of the general practitioner, and precisely the difficulty of the office consultant specialist as we usually see him. In passing, it may be noted that one of the reasons for the augmenting vogue of group practice by associated specialists is the demand for a more globar and compre-

Digitized by Google

hensive view of each patient. Nevertheless, it must be conceded that even group practice often leaves the patient a *dividual* (as old scholastics said) rather than an *individual,* and that the studies made are apt to remain on an analytic basis rather than aspire to the higher terrace of synthetic work. Otherwise put, the *dividual* is considered to have flat foot for the orthopedist, astigmatism for the ophthalmolgist, gastroptosis for the surgeon, and so on: the *dividual* patient remains a congeries rather than a collection of symptoms. This is a quarrel which the general practitioner and the psychiatrist lunge in common against specialized medicine.

Perhaps no more need here be said of the community of views and tasks between general practitioners of medicine and specialistic practitioners of psychiatry. A presupposition of my argument is that the general practitioner, if he is to live up to the designation "general," ought to grasp and use relatively as much in any one specialty (e. g. dermatology, ophthalmology, psychiatry) as in any other specialty. But, as the general practitioner is ready to agree, he certainly does not know as much about psychiatry as about many other specialties. How shall he proceed?

In the first place, there is often a bit of an obsession to rationalize. The general practitioner feels his education in psychiatry deficient and any post graduate instruction practically impossible. He develops a general *psychopathophobia.* Moreover, if in his presence some psychiatrist inadvertently uses a term like *psychopathophobia,* it becomes easy for the old outcry about terminology to be raised. As a matter of fact, the terms of dermatology or of the chemistry of metabolism are just as difficult as those of psychiatry, often more so. What we are dealing with (to continue with this naive terminological joking) is another phobia, *onomatophobia.* One must, of course, acknowledge that terms for exact and complex things

Digitized by Google

can hardly ever become much less precise, and much more simple than the facts, without destroying the facts. But there are signs that terminology is solidifying and getting less and less equivocal in psychiatry. In any event, the immediate duties of the general practitioner do not carry him into actual mares' nests of terminology.

Suppose the *psychopathophobia* and *onomatophobia* to be sufficiently rationalized. There is another comment on the relation of the general practitioner to psychiatry which has an optimistic tinge. Judging him by the standard of his range in other specialties than psychiatry, the general practitioner is found to be comparatively well up in major aspects of psychiatry.

I will not repeat in detail the arguments before presented concerning the general practitioner's reasonably sufficient equipment for handling the syphilitic, feeble-minded, epileptic, alcoholic, focal brain (that is, these disorders in their major aspects), somatic ("symptomatic"), senile, and even perhaps the psychoneurotic disorders. Inadequate practice, at any rate grossly inadequate practice, is not often found among these disorders, if we judge the practitioner by his achievements in other specialties.

In order to learn something of the status of the general practitioner with respect to psychiatry, I have looked over 500 brief descriptions of mental patients going to the psychopathic hospital in cases of the group we call the "temporary care group." That group is composed of patients who have never passed through the probate court or any judicial procedures. They are cases which physicians, or in some instances the chiefs of police and heads of boards of health, have thought suitable for observation at the psychopathic hospital. There is nothing mandatory about the hospital's receiving these patients for observation. The law says that the hospital *may* receive these cases for observation. Accordingly the admitting officers

Digitized by Google

must make some preliminary observations of their own before the patients are admitted for observation. It must be carefully noted that these patients are *admitted,* not committed, to the hospital. A similar law to that for temporary care in Massachusetts is also in force in the municipality of New York. The law forms, as is well known, the acme of procedures in the interest of the mental hygiene of a community, as the modern psychopathic hospital acts as a magnet to bring psychopathic and all sorts of acute, curable, incipient or dubious cases to the state's observation and care.

For my present purpose, however, the point I wish to bring out is that this clinical material is very similar to and almost identical with material which, in most parts of the country, remains for many weeks or months under the control of general practitioners without recourse to the courts. Something like 1,200 or 1,500 patients of this order are under observation at the psychopathic hospital in Boston during a period of a year. They pass rather rapidly through a hospital of 110 beds, operating at from ninety to ninety-five beds, and are then passed back into the community or forward into receptacles for more chronic cases. Something like one third of all the patients are turned back into the community under proper supervision or, if possible, without supervision. This material, then, is exactly what the general practitioner has to deal with, though in most states he has no recourse to a modern institution like a psychopathic hospital. Accordingly the remarks about these patients made by the general practitioners will probably indicate what the status of psychiatry is in the mind of the general practitioner in Massachusetts.

Of course, among these "temporary care cases" there will be a number which have passed through the hands of specializing neurologists or psychiatrists.

A report by such a specializing neurologist may run as follows: "Emotional deterioration, apparent

Digitized by Google

impairment of memory. Hallucinated, aural. Depressed. Delusions, somatic type. No initiative. Listless, apathetic. Poor cooperation. No capacity for work." Such a report is informatory, suggests the diagnosis dementia praecox, and is a fair sample of the routine report of a neurologist. The idea "emotional deterioration" is in our experience almost beyond the range of the general practitioner, nor have the words any very profound meaning for many excellent internists. Yet it would be comparatively easy in a brief period of observation in almost any hospital for the insane (not necessarily a psychopathic hospital) for a general practitioner or an internist who wished to brush up this specialty to learn the special meaning of a phrase like emotional deterioration.

Another neurologist's report speaks of an army case as "showing marked motor acceleration, flight of ideas, and press of activity." These are phrases due to the effect of the Kraepelinian psychiatry on our American institutions. I am not at all sure that I believe the general practitioner of medicine in America should learn all the slang of the psychiatric clinics; but the general ideas underlying the differential diagnosis between dementia praecox and manic depressive psychosis ought certainly to be understood by every general practitioner.

We also receive many cases referred from general hospitals, some of which are of the highest standard. The only instance in which catatonia was specified in any of these reports came from such a high grade general hospital. The fact that an assistant physician in a general hospital should know of the existence of catatonia and be able to specify it in a report on a case ought not to be a remarkable fact. Still it is an isolated fact in the series of 500 cases whose temporary care data I looked over. We, I believe, did not determine that the catatonia in question really was a catatonia, but that is not the point I make.

Digitized by Google

How many general practitioners are advised of the existence of catatonia and really envisage catatonia as they might sundry conditions in specialties like dermatology?

From a very good general hospital we received a statement something as follows: "Onset sudden with acute retention of paralysis (flaccid) of legs ten days ago. Severe pain in lumbar region preceding above. Temperature of 103 to 104. Remains high. Mentally deranged for about twelve hours." Mental derangement! This is a phrase like several others—"mental failure," "irrationality," "mind unbalanced," "cerebral degeneration," "nervousness," "patient erratic"—which phrases all seem to dodge the specific issue and to argue an utter lack of specification in the mind of the practitioner as to different forms of mental diseases and symptoms. There will be found in these reports excellent detailed accounts of the radiation of pain, the situation of tenderness, the Wassermann reaction, the phthalein test with digitalis, the appearance and disappearance of edema of the ankles, and the like; but there is no corresponding objectivity in the account of mental symptoms.

To our astonishment, we one day received from a high-grade general hospital the following: "Patient has been moderately depressed. Yesterday and last night patient became confused, showed fleeting hallucinations and ideas of a somatopsychic character, somewhat nihilistic. Unreasonable. At times disoriented. Difficulty in keeping the patient in bed or on the ward." We afterward discovered that this report had been made by one of our own former psychopathic hospital interns who had become connected with the staff of the general hospital in question. Here was a very informatory report, but one apparently quite beyond the range of most hospital practitioners.

Let us now come to the general practitioners themselves. There is a group of reports in which absolutely nothing is said of value. For example: "A

Digitized by Google

violent and dangerous maniac" *seems* to say a good deal about a patient, who, however, turns out to be neither maniacal, dangerous, nor violent. Another statement, "This man has been acting queerly for the last six months and seems to be all run down," is of little value, as also the report, "Has been gradually becoming mentally weak and physically weak." With all due allowance for the carelessness of some practitioners and the fact that many things may be said in conversation that cannot be put on official slips, it is clear that there is a good deal of dodging the issue, and what in the military service was so universally known as "passing the buck," in the field of general practice as it touches psychiatry.

On the other hand, there are numerous perfectly objective reports which, though they indicate absolute ignorance on the practitioner's part concerning psychiatry, nevertheless are very informatory, for example, "Crying and laughing. Breaking furniture. No sleep for three nights. Very destructive. Burned $150 in bills. Threw watch and chain away. Burned up new shoes and clothes." There is a report which tells something, though from other evidence we are aware that the physician in question has no claim to psychiatric knowledge.

Here is another objective report: "Patient does not sleep, is depressed, is afraid she is going insane, and has expressed a desire to die, but has never spoken of an attempt at suicide. The patient says she is worried about something in the past which she can never tell any one. Patient has an irregular heart, possibly on an arteriosclerotic basis."

Or take another objective report: "This man sits and broods, avoiding the company of other people always. He will not enter a street car if others are present. If empty, he will probably go in and sit down. He says other people have no use for him. He imitates actions of others in minute detail. He says he can't go on living this way."

Digitized by Google

These objective reports indicate clearly that we have in many practitioners a great deal to build on in our propaganda for the extension of psychiatric knowledge among general practitioners.

I have collected a number of instances of the grossly inaccurate use of phrases. Sometimes the users of these phrases are otherwise objective enough. Their inaccuracy merely points to their inadequate medical education. They are perhaps good observers, but poor reporters.

For instance, the statement that a girl has "hallucinations that a fellow has moved into the same house with her" means in modern technical phrase not "hallucinations" at all but "delusions." This confusion of hallucinations and delusions is the most common error in these reports. You may regard it as a venial error, seeing that as a rule the context will tell the psychiatric examiner whether the reporter meant delusions or hallucinations. That is of course true. But my point is that this confusion argues elementary ignorance on the part of the general practitioner, since practically the first thing which a medical student learns concerning psychiatry is this distinction between hallucination and delusion.

A more gross error in phrasing is the following: "Dementia resembling circular insanity developed." The fact that circular insanity, even if the phrase were now in common use, is a nondementing psychosis and does not resemble a dementia ought to be common knowledge among practitioners.

Such ignorance as this can readily be repaired by postgraduate work, and it is clear that a propaganda to this end will meet general approval.

It will be more difficult to eradicate odd ideas concerning etiology. In answer to the question, "What is the cause of the patient's mental derangement?" facts from the anamnesis are almost at random picked for exposition. Thus, "Patient gave her wrong age

Digitized by Google

in securing a position and said that she had two brothers in the army. She feared she would be found out in these lies and arrested." Another, to the question, "What is the cause of the patient's mental derangement?" is *"in love?"* Another, "Overstudy." Another, "Has worked very hard for the past six months." These facts on examination seem to be merely items in general situations. No one nowadays denies the psychogenic origin of a certain portion of mental disease or the psychogenic factor in every mental disease, but we must certainly do something about our statistical blanks if we are not to perpetuate fantastic etiology in the common run of cases. A patient, who is described as having "fallen forty years ago and injured his back, excited, destructive, depressed," has as the cause of his derangement "high blood pressure." This patient turned out to be a case of hemorrhagic encephalitis. Another sample: "Patient comes in after an argument over something. In this case it was over the proper way to fix the bent stern of a submarine chaser which he was working on, and he being a house carpenter undertook to make the ship carpenter do it his way, hence the attack."

I need make no further comment on these etiologic statements. An extension of our propaganda to the demonstration of a few samples of the major groups of mental disease in clinics for general practitioners given at the various hospitals for the insane nearest the centers in which general practitioners might congregate would no doubt serve to erase most of the etiologic rubbish here hinted at. But the propaganda would also wipe out some of the grossly inaccurate use of phrases above mentioned.

Meantime the internists and general hospital physicians would have to brush up their psychiatry and undergo a reeducative or an educative process. All of which would minister to the mental hygiene of the community.

Digitized by Google

SUMMARY

Psychiatry has become almost more popular with nonmedical mental hygienists than the medical profession. Of course, the relations that are ultimately to stand between clinical neurology and psychiatry are not entirely clear. But the relations between psychiatry and the general practice of medicine are disturbed by special difficulties, e. g., phobias on the part of the general practitioner concerning nomenclature and concerning his own supposed ignorance of psychiatry.

A frontal attack is proposed on the general practitioner, in addition to the flank attacks considered desirable in the past, for his proper postgraduate education.

Psychiatry is more a synthetic art than is clinical neurology, now predominantly analytic. But, being synthetic, psychiatry has much in common with general medicine. General medicine, psychiatry, and (to a certain point) obstetrics treat the patient as an *individual,* whereas the majority of the specialties treat the patient (in scholastic phrase) as a *dividual.*

The body of the text contains material illustrative of some inadequacies of the general practitioner *re* psychiatry. Many of these are easily reparable.

ABSTRACT OF DISCUSSION

Dr. E. Stanley Abbot, Belmont, Mass.: Dr. Southard has advocated a frontal attack on the general practitioner. I think we should also make an attack on the medical student. After all psychiatry deals much more with abstract than with concrete symptoms. Not many men are interested in abstract things, but the psychiatrist has to be. We want to get hold in the schools of those medical students who can be interested in abstract topics of conduct, action and so on. In order to do that we must begin earlier than the subject of psychiatry is usually taken up in the medical schools. There has been difficulty in interesting medical students in psychology, partly because of the lack of development of the science itself. Until very recently it has not dealt with the every day matters of conduct and behavior, and yet that is what the psychiatrist is constantly running up against. Psychology as taught seems to have had no relation to

Digitized by Google

psychiatry. What we need, therefore, is the introduction into the curriculum of the medical schools of a psychology that deals with the mental activities involved in the normal adjustments of everyday life and their application to the behavior disorders that are the subject matter of psychiatry. A few years ago I looked up the number of medical schools teaching psychology and there were only twelve or thirteen that had any place for it in the curriculum. I think that is one of the reasons why the general practitioner has so little knowledge of psychiatry when he goes into practice. Psychology seems to the average man an abstruse and mysterious subject with which he is incompetent to deal. That attitude must be broken down by avoiding the language and terminology that is so mystifying to him. By presenting a psychology of every day life as a preparation for psychiatry more men will be interested in it and go out as general practitioners without the defects of special knowledge which Dr. Southard deplores.

Digitized by Google

AN ORIGINAL TEST FOR THE PATHOLOGIC GREAT TOE SIGN

WITH ILLUSTRATIVE CASES

LEO M. CRAFTS, B.L., M.D.
MINNEAPOLIS

While making a study of a remarkable grouping and diffusion of various reflex responses found in the followng case of back injury referred by Dr. J. F. Norman of Crookston, in May, 1917, my attention was attracted by one among other highly interesting phenomena, to a sharp dorsal extension of the the great toe occurring when an upward stroke was made with a blunt point over the anterior surface of the ankle. Application of this test in various cases following this observation led to the definite conclusion that an original method of eliciting the pathologic toe sign had been discovered.[1]

REPORT OF CASE

CASE 1.—T., a single man, aged 52, while reaching under a section of barn floor which was being lifted by several men, was doubled over sharply when they accidentally let it down on his shoulders. He felt a band of numbness around the midbody immediately, but continued his work. For three months he suffered from a dull pain in the region of the original numbness. This gradually improved, and was followed by sharp "cramplike" pains in the same zone. These subsided slowly and were succeeded by numbness in the feet, gradually rising to the waist line. About three weeks later the legs began to grow weak, complete loss of motion rapidly supervening, followed immediately by retention of urine.

The patient came under observation about four days following the onset of these symptoms. His general nutrition was poor, and he had lost 30 pounds. The face muscles and upper extremities were normal, but there was marked

1. Preliminary material on the subject was presented before the Minnesota Neurological Society, Jan. 15, 1918, and an abstract published in Minnesota Medicine 1: 110 (March) 1918.

Digitized by Google

atrophy in the legs. The patellar response was abolished on both sides. There was no ankle clonus, but a marked Babinski response in each foot, and after an interval of about a second extension of the other great toe, accompanied by contraction of the adductors of the thigh. There was also positive reaction to the Oppenheim and Chaddock tests in both feet. Moderate dulling of tactile and pain sense was found over both lower extremities up to a point 2 inches below the umbilicus, and of tactile sense only to a line 2 inches above this point. When the pin point passed the lower of these lines, in determining its topography, it was noticed that dorsal extension of the great toe occurred, accompanied by retraction of the leg. All motor power was abolished in both legs. The abdominal reflexes were obliterated. During the investigation of these various phenomena it was accidentally noticed that stimulation of the anterior surface of the ankle was uniformly followed by dorsal extension of the great toe. And various tests showed that the best response was produced by an upward stroke with a dull point, for a distance of about 2 inches.

A laminectomy was performed by Dr. H. B. Sweetser, May 5. The ninth to the twelfth spines were removed, exposing a brownish, fusiform, extradural mass opposite the ninth and tenth vertebral bodies. This was carefully removed and found to be a hypernephroma.

May 10, the patient reported his legs feeling more comfortable. The Oppenheim and the Shaddock responses had disappeared. The Babinski reflex was present on both sides; and an upward stroke over the ankle surface also produced extension of the great toe. Sensation had improved in the segmental zone below the umbilicus, and that just above it had become hyperesthetic.

May 28, it was reported that the zone of hyperesthesia had disappeared about two weeks before. Sensation was normal now to the line 2 inches below the umbilicus; but below this point there was no change. As the pin passed this line there was still an occasional extension of the great toe. The Babinski reflex was present on both sides, and muscular contraction of the shoulder and of the upper arm of the same side followed the test after about one second's delay; but diffusion did not now cause extension of the opposite great toe. Stimulation of the skin over each ankle also produced extension of the toe, and there was a slight Oppenheim on the right. Patellar response was absent, and no ankle clonus existed. There was no control of the bladder and no power in the legs. The patient returned to his home at this time.

About eight months later he reported continued improvement, with power to reflex and extend the legs, move the toes and to lift either foot from the floor. And he was then able to get from his bed to his wheel-chair and back alone.

Digitized by Google

COMMENT

All the various methods that have been devised by different observers for eliciting the great toe reflex, Babinski, Oppenheim, Gordon, Chaddock and that which I have described, are of identical significance, predicating definite involvement of the motor neuron at some level. The explanation of the occurrence of a positive reaction to one of these tests and its absence to others, in any given case, is an interesting study, and the reason evidently depends on the exact level of arc, or on the individual sets of fibers involved, one sensory stimulus linking with and another missing the activation of the exact motor path concerned. In properly bringing out the various responses, the skill of the technic employed is of distinct importance. All require deft application for accuracy and certainty of conclusion. To develop the Babinski response, Barker directs stroking with a blunt point along the inner plantar border. This is certainly not the best practice. The surest reaction is always found by stroking nearer the outer margin or across the ball of the foot. Sometimes it will be found only by stroking back near the heel; again, midway forward, and in many, only by a stroke across the ball of the foot inward. At times a very light touch, at others deep and firm pressure, produces the best response. A quick stroke may be most effective in one instance, a slow movement in another. All should be employed fo rexact and complete knowledge. The clumsy rubbing of the thumb nail along the sole of the foot is extremely crude. A blunt-pointed instrument properly shaped for the purpose should always be employed for the Babinski test and for mine. Oppenheim's sign is best determined by running the soft ball of the thumb or finger tips, with firm pressure, down the lower 3 inches of the inner posterior border of the tibia. A similar movement around the external malleolus from behind forward brings out the Chaddock. And a quick, deep grasp of the calf muscles develops Gordon's sign. The best method for eliciting

Digitized by Google

the reflex response under present consideration is by stroking with a blunt point upward over the dorsal surface of the ankle, for a distance of about 2 inches, the lower leg being horizontal and the musculature relaxed (Fig. 1). The amount of pressure necessary varies in different cases. A direct, quick thrust against the surface with the point of the instrument is sometimes effective, and a downward stroke occasionally also, but not as uniformly as the upward movement. Usually the test finds the best response when applied up the median line (Fig. 2), but occasionally it will be found either toward the inner or the outer side of the ankle surface.

Of all methods of developing the positive toe sign, Babinski's is undoubtedly most constantly effective. Of the others, I have found my method next in constancy, and distinctly more frequent than that of Oppenheim, while a response to either the Chaddock or the Gordon test is decidedly uncommon in my experience. To be clearly positive, a dorsal extension of the great toe is essential. Simple immobility of the toe is atypical, but its meaning or importance is not certain. The behavior of the other toes varies widely. They may flex, extend or remain inert; or they may spread, fanlike. This behavior of the small toes should have more careful observation for possible added value in the complete interpretation of these tests.

While a considerable series of cases has been under observation and study in determining the value of this test, it is not deemed necessary or desirable to extend the text by detailing numerous case histories. Brief presentation, however, of one or two striking illustrations of its presence in chronic degenerative and acute inflammatory conditions may not be amiss.

ILLUSTRATIVE CASES

CASE 2.—H. K., a man, aged 40, referred by Dr. C. M. Kistler, had had a chancre fifteen years before. He was a plumber by occupation, had been married twelve years, and of his seven children four were stillborn. He had pain between the shoulders and down the back for two years, and

Digitized by Google

increasing trouble in controlling the urine. There had been a loss of sexual power in the preceding two months, and steadily increasing difficulty in walking; but he was in good nutrition and color. The right pupil was larger than the left and not circular; it did not respond to light. The iris had that tense, flat appearance with loss of the normal velvety softness, as characteristic of the tabetic eye as the so-called Argyll Robertson pupil, which I first described[2] in 1917. There was moderate incoordination in the arms, and all arm reflexes were abolished. Patellar responses were entirely wanting. There was decided incertitude of gait and marked ataxia of station. No ankle clonus appeared, but marked Babinski, Oppenheim, Chaddock and Gordon reflexes were present, and a positive response to a stroke over the dorsal surface of the ankle on both sides. Serologic findings in the spinal fluid were positive; in the blood, negative.

Case 3.—Paul, P., aged 7, seen in consultation with Dr. G. M. Doran, Feb. 22, 1919, presented an unusual picture of acute bulbar paralysis (lethargic encephalitis), apparently of influenzal origin. The mother was of a neurotic temperament. About four weeks before, following a rather slight indisposition, probably influenzal, tendency of the eyes to turn in was noticed. The tonsils were removed a week later. Crossing of the eyes increased and became continuous. Two weeks later the child began to have distinct difficulty in speech and had several attacks of vomiting. About five days afterward difficulty in walking developed, accompanied by marked staggering. Swallowing was not involved at that time. When first seen he was pale, and there was a marked internal strabismus, double, but with greater involvement of the right eye. The pupils were normal; speech, very dysarthric, and there was slight power of phonation. The facial muscles were all in action, but the tongue did not protrude. There was some incoordination in arm movements, pendulum swaying when he sat erect, a reeling stagger when on his feet and inability to stand without support. The arm reflexes were negative; the patellar response, overactive and equal. There was no ankle clonus. The great toe sign was positive to all tests, including that which I have described. Distinct lethargy. No nuchal rigidity existed, and no Kernig sign. The next day swallowing was affected. Respiration became difficult; the heart, weak; the pulse, faint and fast, and, following a rapid failure of strength, the patient died that evening.

SIGNIFICANCE OF RESPONSE

The commonly accepted interpretation of a dorsal extension of the great toe on stimulation by any of the

2. Crafts, L. M.: The Early Recognition of Multiple Sclerosis, J. A. M. A. 69: 1130 (Oct. 6) 1917.

Digitized by Google

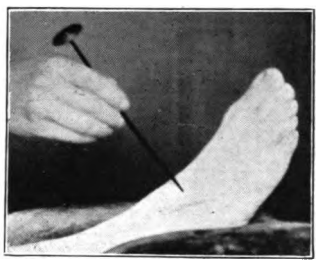

Fig. 1.—Method of eliciting test: blunt point in position for beginning of stroke.

Fig. 2.—Typical dorsal extension of great toe in the application of the test.

Digitized by Google

Digitized by Google

various tests is the presence of definite organic damage to the motor tracts or fibers at some level. And, undoubtedly, this is usually the condition; but not always. I have found the reaction present with the very earliest symptoms of acute cerebrospinal meningitis, on the second or third day in several cases, before organic destruction of motor neurons could have taken place; and I have seen the reaction diminish and entirely disappear with advancing recovery, no motor disability remaining. Barker's statement, therefore, that the positive toe sign does not appear until the second week is not correct.

SUCCESS OF NEW TEST

During the past two years, in which I have studied the occurrence of the pathologic great toe sign by the test here presented, it has been demonstrated in a fairly wide range of conditions of traumatic, inflammatory and degenerative type, including multiple sclerosis (frequently), spinal cord tumor, brain trauma, cerebral apoplexy, brain tumor (both cerebral and cerebellar), brain abscess, symptomatic epilepsy, syphilitic myelitis complicating tabes, spinal cord trauma, and lethargic encephalitis. It would therefore appear to be established as comparable to the Babinski test, only second to it in constancy of occurrence, and of identical significance and importance.

ABSTRACT OF DISCUSSION

Dr. George A. Moleen, Denver: The last word has not been said about the reflexes as indicating destruction of the pyramidal tracts. We have all been impressed with the variety of stimuli applied to the leg which would produce an extensor response in cases of organic lesion of the projection tracts. Even the pricking of a pin, in such frank degenerative conditions, has produced extension of the toe, and the more severe the stimulus the greater the associated response. I think not only the dorsal foot reflex but the von Bechterew, the Babinski, the Gordon and the Oppenheim have a somewhat similar interpretation for most of us and must be accepted as indicative of a lesion, more or less

Digitized by Google

destructive or inhibitory, of the tracts. In a recent case in which all the reflexes were lost, as well as all function below the region of the ninth dorsal segment and after a laminectomy, the recovery of partial function was evident. Most interesting was the reflex to which Marie has called attention, the reflex of inhibition, consisting of a voluntary flexion of the thigh on forced plantar flexion of the toe. This reflex seemed to be an elaboration of that which we have regarded as the more elaborate form of a Babinski reflex. I believe it is yet to be decided which of these reflexes is to be considered as indicative of partial lesion, and which a complete, of the spinal cord, and meanwhile hope that something more definite will be brought out besides the association of other reflexes on which we must all rely at the present time.

Digitized by Google

A CASE OF TUMOR OF THE PONS VAROLII

TOM BENTLEY THROCKMORTON, B.Sc., M.D.
DES MOINES, IOWA

History.—A white lad, aged 17½ years, first came under my observation in October, 1912, complaining of nervousness and inability to walk properly, conditions which were said to have been present for a number of years but to have progressively grown worse during the previous three or four years. The family and personal histories were negative. The onset of the trouble had been insidious. At about 8 years of age, it was first noticed that the boy's right eye had a tendency to turn inward. This was progressive, so that at the end of five years complete internal strabismus was present. Vision and gait, however, could not have been greatly interfered with at the time, for it was ascertained that the lad won an egg race at a town carnival, the winning necessitating the transference of a number of eggs from one spot to another, dipping them up and carrying them by means of a spoon. An itinerant eye specialist attempted to overcome the visual deformity by doing a tenotomy on the internal rectus muscle. Then because the eyeball appeared more prominent and the lids were more separated on the right side, an endeavor was made to decrease the palpebral fissure. In other words, it would not be unreasonable to assume that the ocular condition at that time consisted of a complete paralysis of the external rectus, proptosis and incipient seventh nerve weakness. The next symptom was weakness of the right face, followed by diminution of hearing, first on the right and later, to some extent, on the left side. For a number of years it was stated that the lad had been uncertain in his gait, but that this condition had grown much worse during the previous three or four years. The inability to handle himself properly was evidenced by scars, recent bruises and cuts, which were present at the time of examination, about the tibial regions, being self inflicted in an endeavor to use such objects as an ax, corn knife or other implement. For six or seven years, headaches had been complained of intermittently; but the family attributed the cause to "eye strain," and later, when vomiting accompanied the cephalalgia, "biliousness" was thought to be the causal

Digitized by Google

factor. It was learned, however, that the vomiting occurred at times independently of the ingestion of food and not infrequently was of the projectile type.

Examination.—The gait was reeling, lurching and staggering, with a marked tendency to veer more to the right. The ataxia was of the cerebellar type, not being appreciably influenced on closure of the eyes. The so-called characteristic cerebellar attitude of the head was present; that is, the chin turned to the affected side and the head tilted to the contralateral shoulder. A double choked disk existed, more marked on the right side, and the visual acuity on this side was 6/60, as compared with 6/10 on the opposite side. There was slight involvement of the ophthalmic division of the right fifth nerve, as indicated by diminution in corneal sensitiveness, while the sixth, seventh, eighth, ninth, tenth and eleventh cranial nerves on the right side were all involved to a marked degree. Involvement of the left sixth, eighth and twelfth nerves was evident from weakness of the external rectus muscle, moderate diminution in hearing, and a tendency for the tip of the tongue to curve to the left side on protrusion. The latter condition, however, may have been more apparent than real, as the pulling aside of the paralytic right cheek caused the tongue to protrude more nearly in the median line, its curvature to the left possibly being due to the prolonged habit of being displaced by the paralyzed muscles of the right cheek. The motor pathways to the left arm and leg were involved, as shown by motor weakness, increased tendon jerks, hypertonia of the leg muscles, and an extension of the great toe by the Babinski and Chaddock methods. The crossed paralysis syndrome, indicative of pontile lesions, was beautifully illustrated by the seventh nerve lesion of the right side associated with motor paralysis of the opposite arm and leg. The laboratory findings were completely negative as far as the blood Wassermann, subcutaneous tuberculin, urine and blood tests were concerned.

Neurologic Symptoms.—General Symptoms of Brain Tumor: (1) headache; (2) projectile vomiting; (3) double choked disk; (4) vertigo.

Cerebellar Symptoms: (1) ataxia, uninfluenced on closure of eyes; (2) characteristic attitude of head; (3) reeling, lurching, staggering gait, more toward the right; (4) dysdiadokokinesia; (5) asynergy; (6) nystagmus.

Brain Stem Symptoms: Right: Optic, choked disk; O. V. D., 6/60; trigeminal, ophthalmic division; abducens, complete; facial, complete; auditory, almost complete; glossopharyngeal, pneumogastric and spinal accessory, all involved to a greater or less degree; motor pathways to left arm and leg.

Left: Optic, choked disk; O. V. S., 6/10; abducens, moderate; auditory, moderate; hypoglossal (?).

Digitized by Google

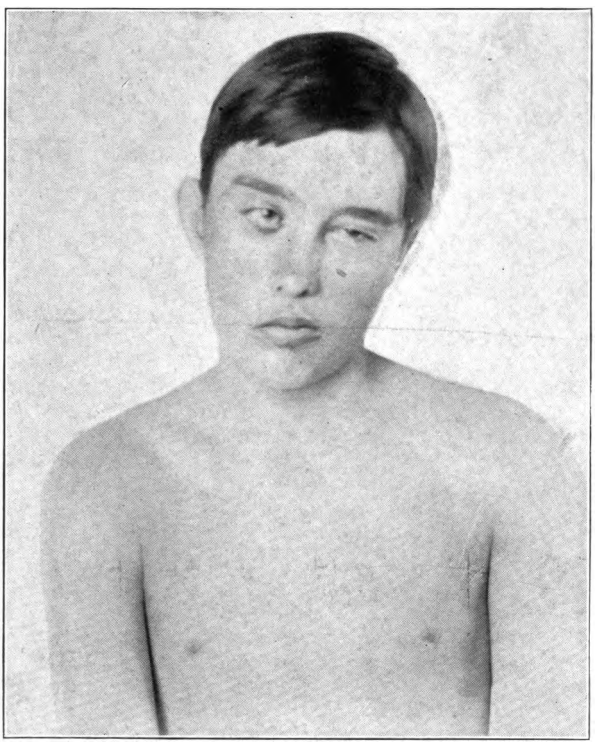

Fig. 1.—Characteristic position of head; also the right sided proptasis, the right sided facial paralysis and the distinct lowering of the right shoulder. The left sided ptosis is more apparent than real.

Digitized by Google

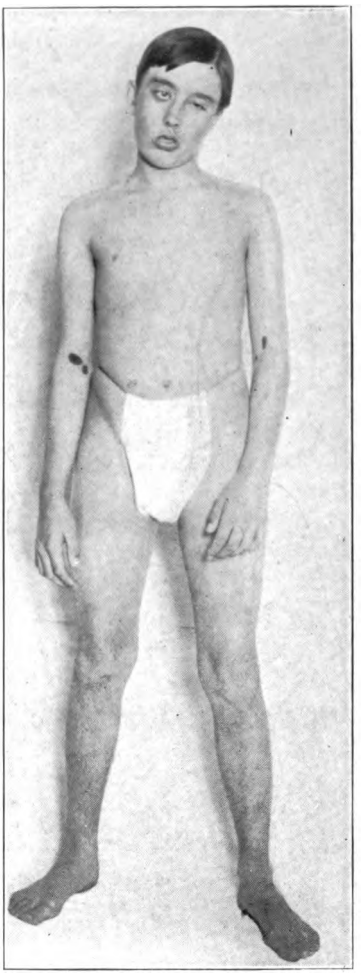

Fig. 2.—Characteristic attitude assumed when standing. Note the broad base obtained by spreading the feet apart, an endeavor to overcome the ataxia (flashlight photograph).

Digitized by Google

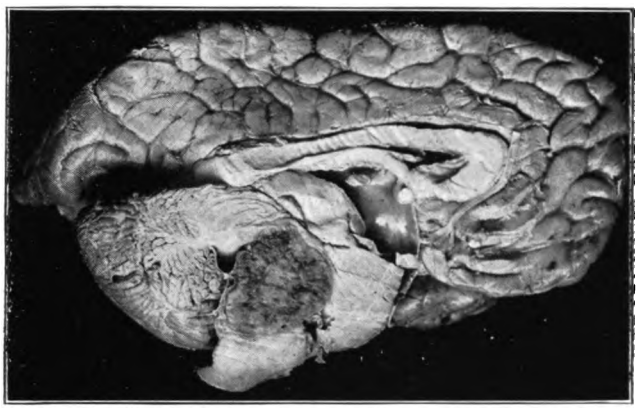

Fig. 3.—Mesial section through brain, showing pontile tumor.

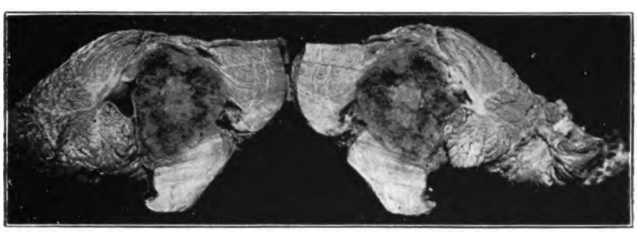

Fig. 4.—Section through brain stem and cerebellum. Note atrophy of right cerebellar lobe.

Digitized by Google

Digitized by Google

Operation and Result.—A decompression operation was performed by Dr. C. E. Ruth, at which time the entire right cerebellar lobe was exposed, first, in the hope of relieving the symptoms due to increased intracranial pressure, and, secondly, that a tumor might be found in some accessible position. Nothing, however, was ascertained as to the presence of a tumor, save an increased resistance, felt by the palpating finger in the region of the pons. Postoperative convalescence was uneventful until the sixteenth day was reached, at which time, without apparent cause, the fever rose to 103 F. unaccompanied by pain or discomfort. A hernial protrusion over the operative site became so marked as to warrant the necessity of a paracentesis, at which time 75 c.c. of clear cerebral fluid were withdrawn. Two days later the eruptive rash of smallpox appeared. Following the recovery from this disease, the patient returned to his home, where he lived in comparative comfort for almost five years subsequent to the operation. During this time his general health greatly improved, he was free from headaches and vomiting, and while a double optic atrophy slowly followed in the wake of the preexisting papillitis, he never completely lost his eyesight. Needless to state, the organic changes in the nervous mechanism produced by a growth, the first symptom of which was observed when the boy was 8 years old and which ultimately caused his death fourteen years later, never were eradicated; but the prolonging of life with comparative freedom from symptoms which would have made death preferable, more than justified the operative interference.

Necropsy.—Sixteen hours after death, a necropsy was performed by Dr. D. J. Glomset, at which time the brain was obtained. The dura mater stripped easily from the calvarium save at the point of the old operative site, where the attachment of the meninges was quite firm. There was considerable atrophy of the right cerebellar lobe; but no external evidence of a tumor could be found, save that indicated by the presence of several pia-arachnoid cysts which lay along the under surface of the brain, particularly at the junction of the transverse fibers of the pons with the medulla. After the brain had been hardened, a mesial section was performed which revealed a spherical mass 3.5 by 4 cm. in diameter, occupying the space between the superior cerebellar peduncles and the crus cerebri above, the medulla oblongata below, the transverse fibers of the pons anteriorly, and filling, practically to its entirety the fourth ventricle. The center of the mass was somewhat necrotic and showed evidence of recent hemorrhage. The tumor was distinctly encapsulated and without great difficulty was separated from its capsule except at the point of its greatest attachment, which apparently sprang from the longitudinal fibers of the pons and their continuation

Digitized by Google

downward through the medulla. The outer surface of the growth was quite irregular and nodular, the nodules penetrating the cerebellar tissue, particularly on the right side. Microscopic examination of the tumor showed that it was gliomatous.

ABSTRACT OF DISCUSSION

Dr. Frank W. Langdon, Cincinnati: I am interested in this case as an example of careful study and accurate localization of a basilar tumor. We should recognize the fact that there are two groups of these cases which come before the clinician, in one of which he advises early operation to save the patient's vision. Exploration which is practically harmless is not only allowable but we are warranted in taking some chances. How far we shall go after that is another matter. But there are two obstacles in the way even to exploration. There is one class of patients who will decline any form of operation, and there is another class in which the general condition is such that operation is not advisable. You are facing what will eventually be a fatal disease, the patient meanwhile being liable to suffering and blindness which may be prevented by early exploration and decompression. We should not rely on hard and fast rules, but in suitable cases explore and be guided by what is found.

Digitized by Google

NEUROLOGIC SEQUELAE FOLLOWING ACUTE RESPIRATORY INFECTIONS *

WILLIAM H. ROBEY, JR., M.D.
Assistant Professor of Medicine, Medical School of Harvard University
AND
HEIMAN CARO, M.D.†
BOSTON

The etiologic factor in the cases cited in this report was difficult to determine and has caused some doubt as to the most appropriate title for the paper. The first case occurred during a period when a small number of cases developed in the camp characterized by a general bronchitis and malaise. The illness was mild, and many cases were so slight that the patients were cared for by the regimental surgeons and were not sent to the base hospital. The initial illness in the first case of the report was apparently mild, and the patient was not admitted until four weeks later, when the neurologic symptoms appeared. The second case appeared during an epidemic which lasted for about six weeks, and existed in several army camps in this country and Europe. For lack of a better name, and because of our ignorance of the etiology, it was called "three day fever." Eight hundred and fifty patients were admitted, and of these 500 entered the hospital between April 12 and April 20. One hundred cases were studied intensively, clinically and bacteriologically.

The characteristic symptoms were: onset with chill or chilly sensations, 72 per cent.; headache, 93 per cent.; general malaise, 92 per cent.; pain in joints and chest, 90 per cent.; cough, 62 per cent.; backache, 51

* From the Medical Service of Camp McClellan, Anniston, Ala.
† Captain Caro died of pneumonia in January, 1919, at Base Hospital No. 107.

Digitized by Google

per cent.; sore throat, 21 per cent., and pain in eyes, 12 per cent. The physical findings were: redness of throat, 51 per cent.; râles scattered through the chest, 50 per cent.; redness of eyes, 11 per cent., and herpes facialis, 5 per cent.

On admission the patients felt very ill, but within twenty-four hours the majority were anxious to return to duty. The complications were slight in the hundred cases studied: bronchitis, 25 per cent.; pneumonia, 1 per cent., and otitis media, 3 per cent. (The clinical data were collected by L. E. Luehrs, First Lieutenant, M. C.)

In the bacteriologic study many organisms were found to be present, but the predominating ones were a nonhemolytic streptococcus in the throat and the nasopharyngeal cultures, while *Micrococcus catarrhalis* was second in frequency. (The bacteriologic studies were made by G. A. Wislocki, First Lieutenant, M. C.)

Involvement of the nervous system as a sequela following acute infections of the upper respiratory tract is by no means uncommon. Mild neuritides and neuralgias following such infections are frequent, and generally present a good prognosis. Neural complications with fatal termination are comparatively rare.

REPORT OF CASES

CASE 1.—*History.*—R. F., a white man, aged 22, was admitted to the base hospital, Dec. 19, 1917, with a diagnosis of "paraplegia, incomplete, and urinary distention."

The family history was negative except that the mother had died of cardiac disease when the patient was quite young. He was of subnormal mentality (moron). He had had measles, diphtheria and scarlet fever in childhood. In 1916, he had an attack of pleurisy involving the left chest. He denied any venereal disease or alcoholic indulgence.

Four weeks prior to admission, he was taken ill with rhinitis, tonsillitis, malaise and cough. At the end of a week he was able to return to his work as a cook, apparently fully recovered. About two and one-half weeks later, or four days prior to admission, he noticed that his left lower extremity was weak and "dragged." Two days later the right was similarly involved, and he had great difficulty in walking. On

Digitized by Google

the succeeding day (prior to admission), he had acute urinary retention and incontinence of feces.

Condition on Admission.—The chief complaint was of pain above the symphysis pubis and inability to walk. Physical examination revealed chronic pharyngitis, hypertrophied and injected tonsils, a small-sized left cervical gland, and scattered, medium moist râles throughout both lungs. Neurologic examination revealed markedly exaggerated knee and ankle jerks, with sustained bilateral ankle clonus, and the Babinski response on the left only. On attempting the plantar reflex, there was a tendency to flex the lower leg ("guard phenomenon"). Pain sensation was diminished from the waist downward, and there were also vague paresthesias. The pupils were equal, regular, and reacted normally. There was no nystagmus nor diplopia.

Course of Illness.—December 21, a complete paraplegia was present. Knee and ankle jerks were absent. From a line midway between the ensiform cartilage and the umbilicus to the toes, pain and thermal sensations were absent, but tactile and muscle sense were preserved. The tendon reflexes of the upper extremities were present and equal. Bladder retention and rectal incontinence continued.

December 25, muscle and tactile sensations were found to be absent in the area described, and there was now complete anesthesia from the ensiform downward.

December 26, complete anesthesia had advanced to the fifth intercostal space, and the patient began to complain of severe epigastric pain. Since his admission to the hospital, his temperature had ranged from 100 to 102 F., always rising at night. The condition of the pharynx and tonsils continued as at entrance. Expectoration became more profuse, but there was no change in the physical signs. There was a flaccid paralysis of the lower extremities, and sphincter disturbance remained unchanged.

December 29, since the foregoing note the patient had become steadily worse. Complete loss of sensation now extended to the clavicles. There was motor paralysis also of both arms. Respiration became Cheyne-Stokes in character. The patient became markedly nauseated, lapsed into coma and died.

Laboratory Findings.—The spinal fluid was under slight pressure and showed 100 cells per cubic millimeter, with as many red corpuscles and a trace of globulin. The Wassermann reaction was negative. The blood count was 10,000 white cells, of which 70 per cent. were polymorphonuclear leukcocytes and 30 per cent. mononuclears. The urine was straw colored and cloudy, with a specific gravity of 1.017, a heavy trace of albumin, no sugar, and a large number of white cells (which may have been due to repeated catheterizations).

Digitized by Google

Necropsy Protocol.—The heart and lungs appeared normal. The vessels along the entire course of the spinal canal were found to be congested, and markedly so over the thoracic region. At the level of the third, fourth and fifth thoracic vertebrae, the cord was very soft and swollen, especially at the level of the fourth, or in the region of the sixth and seventh thoracic nerve segments. Cultures from this region were sterile.

Histologic Examination of the Cord (by Dr. F. B. Mallory).—Blocks of the formaldehyd-fixed cord were zenkerized, and paraffin sections from six different levels were stained by the eosin-methylene blue method. All except the uppermost level showed varying degrees of the same pathologic lesion, namely, large and small areas of degeneration and destruction of the nerve fibers and of their myelin sheaths. In the degenerated areas, the nerve fibers had often entirely disappeared, but in places they were present and much swollen. Where the lesions were at all marked, the remaining tissue (chiefly stroma) was infiltrated with large numbers of endothelial leukocytes, containing vacuoles evidently because of their having been filled with fat and myelin. The endothelial leukocytes occurred diffusely in the tissue and also packed in the walls of the blood vessels. In a few places lymphocytes were present in small numbers, and, in the part of the cord where softening was obvious macroscopically, a few small clumps of polymorphonuclear leukocytes were present, chiefly in the neighborhood of the small blood vessels.

In a section through the lumbar region, the lesion involved the white matter extensively on both sides of the median fissures, and occurred also in foci in some of the lateral tracts on one side. In a section through the dorsal cord, about two thirds of the white matter was degenerated or entirely destroyed. In the softened area, practically the whole cord was involved. Some of the ganglion cells in the gray matter showed more or less extensive retrograde changes, but no necrosis.

Diagnosis.—Transverse myelitis.

Case 2.—*History.*—J. B. M., a white man, aged 26, was admitted to the base hospital, April 7, 1918, with the diagnosis of "acute influenza." The family history was negative except that his mother had died of pulmonary tuberculosis. The patient had had typhoid fever in childhood. In 1912, appendectomy was performed, apparently without drainage. In 1914, the patient spent several months in a sanatorium for pulmonary tuberculosis, but asserted that he was told that he did not have any active lesions. During the months of February and March, 1918, he had two attacks of severe, crampy pains in the muscles of his arms and legs, lasting but a few minutes each time. He denied syphilitic infection. From the

Digitized by Google

age of 20 to 24 he had been very intemperate, he said, but for the preceding two years he had been a total abstainer.

Four days prior to admission (April 3), the patient became ill with photophobia, headache, nausea, vomiting and general malaise. He continued at his duties until the day prior to his admission, when he felt much worse and had a very irritating cough. (His symptoms were similar to those of an epidemic of acute respiratory infection, called influenza, then rife in the camp.)

Condition on Admission.—He complained chiefly of the symptoms mentioned above. Physical examination disclosed a few scattered râles throughout both lungs, but especially at the right base posteriorly. His temperature was 101.4 F. Neurologic examination was essentially negative.

Course of Illness.—April 10, the patient complained for the first time of weakness, numbness and inability to extend his legs. There was a slight tenderness on pressure over the nerve trunks. The knee jerks were absent, but the ankle jerks were present and equal. The normal plantar reflex was present. There were no Chaddock nor Oppenheim signs, and no sensory disturbances. The temperature was normal, and continued so throughout the course of the disease.

April 19, the condition had slowly grown worse. The patient had considerable nausea and hiccup, which were slow in clearing up under dietary measures. Neurologic examination revealed ankle and knee jerks absent, normal plantars present, no ankle clonus, abdominal reflex absent, and the tendon and bone reflexes of the upper extremity present and active. No changes in sensation were noted except that deep sensibility was increased, and that Laseque's sign was present. Vague annoying paresthesias in the form of intermittent crampy muscle pains caused considerable discomfort. They were confined to the peroneal and anterior tibial muscle groups. The patient could adduct and abduct the thigh, but flexion of the thighs or extension of the legs was impossible. There was no toe drop. To faradism, the quadriceps extensors gave no response, while the calf and hamstring groups reacted slowly. The muscles of the upper extremities reacted well to the current, but the hand grips were weak.

April 26, the patient continued to grow worse. Anorexia was now practically absent. The inability to extend the forearms and the weakness of grasp steadily increased. The patient complained of blurring of the vision as regards distant objects, but ophthalmologic examination failed to reveal any process involving the retina, media or eye muscle balance. There was no facial paralysis. There was a slight huskiness of the voice, and examination of the larynx disclosed a weakness but not a true palsy of the vocal cords. The accessory muscles of respiration were not affected. The triceps reflex was absent on both sides; the other upper arm

Digitized by Google

reflexes were diminished in intensity. To faradism, the triceps muscles of neither arm reacted at all. The abdominal reflex was absent, but the cremasteric was present. There was no change in the condition of the lower extremities or in the sensory reactions. There was no bladder nor rectal symptoms. A slight tenderness was present on pressure over the sciatic, the anterior crural, the internal cutaneous (of the thigh) and the musculospiral nerves at their points of emergence on both sides. At this time these nerves appeared to be chiefly involved with the sciatic to only a slight degree.

April 30, the condition of the extremities remained as described in the previous note. There were no sensory disturbances except the crampy pains mentioned. The accessory muscles of respiration showed no involvement, but the breathing had become slightly irregular and labored. The patient complained of pain in the chest and of sensations of constriction. Examination of the heart and lungs detected nothing abnormal. The pulse, however, had increased from 96 to 120.

May 3, respiratory difficulty gradually increased, and evidence that the diaphragm was involved was quite evident. On the day preceding, the temperature rose to 101 F., and there was evidence of a beginning terminal pneumonia. The patient died from respiratory paralysis plus the toxemia of the pneumonic process, the sensorium being clear to the end.

Laboratory Findings.—The urine was clear and had a specific gravity of 1.027. It contained no albumin nor sugar; the sediment was negative. The spinal fluid was under normal pressure, 1 cell per cubic millimeter, with a very faint trace of globulin. The Wassermann reaction was negative. Blood examination revealed that white cells, April 8, were 11,300 per cubic millimeter, with 73 per cent. polymorphonuclears, 4 per cent. transitionals, and 23 per cent. mononuclears; April 12, 7,800, with 63 per cent. polymorphonuclears, 7 per cent. transitionals, and 30 per cent. mononuclears; April 24, 9,200 with 70 per cent. polymorphonuclears, 6 per cent. transitionals, and 24 per cent. mononuclears. Red cells were 5,600,000 per cubic millimeter.

Necropsy.—A detailed report cannot be given, but no gross lesions of the brain or cord were found. Dr. Mallory examined the cord histologically but was unable to make a diagnosis.

CONCLUSION

The first was a clean-cut case of acute ascending myelitis, in which the only etiologic factor appeared to be an acute respiratory infection antedating the neurologic symptoms by nearly three weeks, but which had never entirely cleared up, as evidenced by the chronic

Digitized by Google

pharyngitis and the infected tonsils still present. The second case was more difficult to diagnose, although the etiologic factor was undoubtedly a respiratory infection of unknown origin. We have already shown how varied were the bacteriologic findings in the group of cases to which the second patient belonged.

Of interest is the very selective action of the toxin—the nerves supplying the extensor muscles of the body being more especially involved, while, peculiarly, the wrist-drop and the toe-drop were absent. The picture from day to day was suggestive of an acute Landry's and, later, perhaps of some form of the muscular dystrophies; but, the symptoms taken en masse, the diagnosis of acute polyneuritis seems unquestionable. The fact that such complications may follow comparatively mild infections of the upper respiratory tract is a matter that always warrants grave consideration and, as is well known, a very guarded prognosis.

We are greatly indebted to Dr. F. B. Mallory of the Harvard Medical School for his histologic studies of the cords.

ABSTRACT OF DISCUSSION

Dr. Charles A. Rosewater, Newark, N. J.: I would like to ask if Dr. Robey has heard of cases of sudden death following influenza or three days fever. In some of these cases the illness was so mild that the patient was not even confined to bed, and yet these individuals died suddenly. A man about 50 years of age awoke at midnight and complained of pain in the heart; he died before he even had a chance to take a drink of water. Inquiry revealed the fact that he had been slightly indisposed for one day a week or so previously, although he did not stop work. Two weeks after the death of this man his 22 year old daughter dropped dead after eating Christmas dinner. No history of previous illness could be obtained. I have heard of other cases of sudden death following the influenza epidemic. An ophthalmologist informed me that he had seen several cases of strabismus following influenza. This may be a condition similar to post diphtheritic paralysis, and it is not unlikely that there are more of these cases than have been reported.

Dr. W. H. Mayer, Pittsburgh: There is no question as to the secondary manifestations of these respiratory affections. There is also no question that the epidemic of influenza was one which may or may not have been due to the

Digitized by Google

influenza bacillus. But many other organisms were present and the lesions of the nervous system which followed were colored by the predominance of this or that type of toxin. I am prone to regard the cases of sudden death, to which Dr. Rosewater referred, as being due to cardiac collapse. It has long been recognized that no matter how mild the primary infection is, the myocardium always suffers, and in my experience those persons who were profoundly ill and who had a long convalescence, especially after influenza, suffered less than those whose illness was of a shorter duration and who had insufficient rest in bed. All manner of mild nervous states with fatigability, in some cases approaching what has been called the neurocirculatory asthenia syndrome, were present. I believe that in these cases the disturbance is primarily cardiac. I realize that I am treading on dangerous ground when I attribute the past epidemic to the influenza bacillus although the work of Klotz and others seems to have satisfactorily established this point despite the presence of the nonhemolytic streptococcus which they have shown to be a secondary invader. By its close association with the influenza epidemic, I regard the epidemic encephalitis as in all probability due to the causational factor of the primary infection. That these cases all present the pathology as so clearly outlined yesterday by Dr. Bassoe I cannot believe since many of the cases of rather complete involvement have cleared up too suddenly. I especially remember one case in which there was a bilateral third nerve palsy and a bilateral seventh nerve involvement. Curiously, there were exaggerated reflexes and a paralysis of a spastic type in the right arm, with absence of reflexes in the right leg, and in the left leg an exaggeration of the reflexes, with normal reactions in the left arm. This condition persisted for several weeks and then recovered quite rapidly. There are many phases of nervous involvement which accompanied the epidemic of last year, whether called influenza or not, which were not peculiar or different from what has been observed in former epidemics except as regards the encephalitis. There is still, perhaps, some question to discuss concerning the reason for the severity of brain stem symptoms which involve lethargy and stupor which occurred in this last epidemic and are different from the involvement of the nervous system as witnessed in former epidemics.

Dr. Max H. Weinburg, Pittsburgh: Like Dr. Mayer I am inclined to connect these two cases with encephalitis lethargica. particularly the first case, in which there was an increased cell count in the spinal fluid. It would seem that these two cases belong to the polyneuritis group of encephalitis which was reported in the Local Government Bulletin of London, No. 121. Unlike the investigators of Europe who state that there were no changes in the spinal fluid in the cases in New York, we found that a great

Digitized by Google

majority showed an increase of cells, mainly lymphocytes. We have found as high as 400 to 600 cells per cubic centimeter. It would be interesting to know whether the other cases in this series have shown other symptoms, such as ocular or facial palsies.

Dr. Alfred Gordon, Philadelphia: Apart from cardiac organic disease, sudden death speaks very strongly of a sudden lesion in the mesencephalon. In the recent influenza epidemic I had an opportunity to observe a very large number of cases with a great variety of neurologic symptoms. I grouped under the name encephalitis lethargica all cases presenting besides the somnolence peripheral nerve involvement in which all the cranial nerves were more or less affected. It would seem that the cases reported by Dr. Rosewater and others belong to one category, namely, disease of the mesencephalon. As to the anatomic basis for it we must refer to Marie's work in which he speaks with great emphasis of the involvement of the gray matter around the third and fourth ventricle, also of the degenerative changes of the locus niger. As to the somnolence reported in encephalitis lethargica, there is nothing especially new about it as this affection has been known many years. In Raymond's clinics reference is made to a large number of cases following infectious diseases, such as paratyphoid fever and pneumonia. Gayet long ago referred to somnolence as being associated with a lesion in the mesencephalon, and he wanted at the time to describe a special somnolence center. By analogy we must draw the inference that all these cases, whether reported as encephalitis lethargica or following influenza and other infectious processes, belong to one category with variations.

Dr. William H. Robey, Jr., Boston: As to the cases of sudden death, referred to by Dr. Rosewater, we did not have any. I do not know how the epidemic of influenza was regarded in this country. In France there seemed little evidence that it was caused by the influenza bacillus. In most cases it was a profound toxic disturbance due to the hemolytic streptococcus. In the experiments of transmitting the influenza bacillus, the conclusions were very unsatisfactory. I believe these were probably due to some toxin. Regarding Dr. Weinburg's question as to the other cases, the only other complication we had was otitis media in a case with pneumonia. Only two patients presented neurologic signs and both died.

Digitized by Google

HYSTERICAL HEMIPLEGIA

REPORT OF CASE RESULTING FROM A SHRAPNEL WOUND OF THE SCALP AND PRESENTING INTERESTING CLINICAL FEATURES

HARRY H. DRYSDALE, M.D.

AND

J. S. S. GARDNER, M.D.

Assistant-Surgeon, United States Public Health Service

CLEVELAND

While the history and clinical picture we are priviledged to report may not be considered especially unusual by those medical officers who, during the great war, encountered all manner of functional nervous manifestations, the condition nevertheless does possess features of interest to the general practitioner and presents several points worthy of discussion by the neurologist.

REPORT OF CASE

History.—W. P. W., a married man, aged 29, American, marine diver, enjoyed good health during youth and obtained a common school education.

So far as we could determine, the family history presented nothing noteworthy. His mother and father were dead; the former died from a complication of diseases at the age of 62; the father succumbed to pneumonia a few weeks before. The patient had two brothers and two sisters. One brother died from wounds, in France, during the past year. The remaining brother and two sisters were alive and well.

April 22, 1902, he enlisted in the United States Navy; he contracted malaria during July, 1903, and was discharged for this disability, May 10, 1904. Shortly thereafter he was awarded a monthly pension of $12, which is still in force.

After this country declared war against Germany, this man applied for enlistment in the military service of the United States, but was rejected at Detroit and Cleveland as "medically unfit," for what reason we are unable to ascertain; but it is presumed that the examining officers took into

Digitized by Google

consideration the fact that he had been discharged from the Navy as physically disabled. May 1, 1917, he was accepted by the authorities at Windsor, Ontario, and went overseas with the Canadian Expeditionary Forces, June 27, 1917.

His service at the front was uneventful until on or about Aug. 1, 1917, when he claims to have volunteered to accompany a rescue party, at night, in search of their captain, who was reported lost in No Man's Land.

The officer was found severely wounded and in need of prompt surgical attention. In the face of a heavy German bombardment this soldier started back to safety, carrying the wounded officer. As he approached the communicating trenches, shells were exploding thick and fast; but he succeeded in delivering his charge to the stretcher bearers. At that moment he felt a sharp sting on the left side of the head and believes he fell forward into the trench. He was treated at the clearing station during the subsequent twenty-four hours and then removed to British General Hospital No. 8, where it was found he was paralyzed on the entire left side of the body, including the face. The period of unconsciousness, he stated, was brief. He was confined to bed for a period of one month. Since then he had been able to get about with the aid of crutches or a cane.

Oct. 28, 1917, he was transferred to England and received treatment and observation in several special military hospitals. During the many examinations he heard his condition discussed as a "hemiplegia" resulting from a shrapnel wound of the left scalp. He intimated that the examiners used the term contrecoup. The weakness of face, he avers, gradually disappeared in a few weeks.

June 7, 1918, he was invalided to Canada and further observed. The disability, however, remained practically unchanged and he was discharged at Toronto, Jan. 2, 1919, as "medically unfit." A monthly pension of $50 was granted.

From the medical history of this soldier as recorded in the final report of the Canadian Medical Board, dated Toronto, Ont., Dec. 12, 1918, a copy of which was kindly furnished me by Major E. E. Fletcher, I quote the following:

"History: Wound of left side of head by piece of H. E. shell and struck on head by piece of timber. This was followed by hemiplegia on same side as wound. Hemiplegia may be due to contrecoup or more likely to some intracerebral vascular disturbance. X-ray of skull negative. Operation considered inadvisable.

"Present Condition (Objective): Power of left arm poor throughout, about one-third normal. Loss of all power for fine movements of left hand. Partial loss of power of left leg. Coarse tremor of left arm and sometimes of left leg. Sustained left ankle clonus. Plantar reflexes give Babinski response on left side. Left foot held in position of partial equinovarus. Can walk (using a stick) with marked limp

Digitized by Google

on left leg. Sensation normal; no astereognosis. Small depressed area on left side of skull about 2 inches above left ear. Is somewhat emotional and at times hysterical. There is slight mental deterioration.

"Present Disability: (1) Partial loss of function of nervous system. (2) Defective eye sight.

"Final Conclusions of Board: Wassermann on spinal fluid and blood negative several times in England. Disability of soldier now 100 per cent. The report also states that this soldier told one of the medical officers there was a weakness of left side of body, following his attack of malaria in 1903; but he now denies having made this assertion."

Examination.—Our first examination, Jan. 20, 1919, disclosed a man of large frame and excellent development. He was exceedingly tremulous, anxious and depressed. He walked with crutches, carried the left arm close to the body, and dragged the left leg in a somewhat flexed position. The fingers of the left hand were firmly contracted but not of the Babinski and Froment type of reflex contracture. A partial left hemianesthesia prevailed; reflexes of the left side were quite sharp; a fairly well sustained clonus of the left ankle and left patella were detected, and as typical and complete a Babinski response of the left foot as we have ever encountered was elicited. The Oppenheim and Gordon signs, however, were absent. The left leg was paretic and slightly atrophied without appreciable change in the electric reactions. No vasomotor or thermic abnormalities existed.

A healed depressed scar, about three-fourth inch in length, adherent to the skull, was found on the left scalp at a point about 2 inches above the left ear. This wound is said to have been infected. The small toe of the right foot was amputated some years ago following a crushing injury.

The heart, lungs, blood vessels and abdominal viscera were normal. The pulse rate was 110, regular and of fair volume. The blood pressure measured 115 mm. systolic and 98 diastolic.

The patient's record, as personally related, plus the physical findings, was strongly suggestive of an organic palsy, and not until his history was further scrutinized and a more careful study made of the symptoms did it appear that we were probably dealing with a functional derangement.

He went on to say that he had returned to Cleveland a cripple, and he became quite emotional as he told of his wife's unfaithfulness during his absence in France and how he had lost his American citizenship by enlisting in the Canadian army. He stated that he had applied at the U. S. Marine Hospital, this city, in the hope that the trouble in his brain might be located and possibly corrected by surgical means. If his condition was incurable he wanted to know it at once, as suicide was preferable to a life of invalidism.

Digitized by Google

Complete roentgenograms of the skull were made by Dr. T. J. Taylor at Lakeside Hospital, and not a single suspicion of fracture was detected.

Dr. Leo Wolfenstein made a painstaking investigation of the eyes, and reported that the patient claimed defective vision of the left eye, apparently about 20/100; but when he placed a plus 6.00 sph. in front of the right eye, the vision of the left eye was found to be at least 20/30. In the right eye, the patient claimed vision of about 20/70, but with a little strategy the vision of the right eye proved to be normal. During his observation of the case the vision seemed to vary from time to time. The eye grounds were entirely healthy, and no error of refraction was detected. The fields also varied considerably from day to day. There was no nystagmus.

The spinal fluid was obtained by Drs. Richard Dexter and C. L. Cummer, and their findings were: Pressure, not increased; globulin, not increased; cell count, none.

The Wassermann reaction read: 0.1 c.c., negative; 0.3 c.c., negative; 0.5 c.c., negative; 1.0 c.c., negative.

Treatment and Result.—Arrangements were then made to subject the patient to a systematic plan of therapy. He was candidly informed that his brain had not been organically damaged, and was assured that if he would cooperate with us a recovery might consistently be expected. It was explained to him that he undoubtedly had been severely shocked and his nervous system undermined thereby; but the trouble, fortunately, was functional.

Sedatives were used to allay the extreme emotionalism and to overcome a persistent insomnia. Saline baths each night, galvanism every other day, and regulated exercises were prescribed.

From the very beginning, a decided change for the better was noted. His nervousness gradually abated, and he became enthusiastic and hopeful. In less than two weeks his gait was very much more stable, and to our surprise the alleged Babinski phenomenon and clonus of the left ankle and knee disappeared. Crutches were discarded for a cane, and within a month he was able to go about unassisted. He complained, however, of difficulty in crossing congested streets. Crowds excited him, and he sometimes feared he might lose his control and be injured by some passing vehicle.

During the first six weeks the left hand remained more or less contracted. He told us, however, that sometimes when alone he was able to walk and use the left arm almost as well as before he was wounded; but the moment he came in contact with people the paralysis would recur. With the use of electricity we seldom failed to relax fully the contracted left fingers, but the result was temporary. As an additional suggestive measure, alcohol was injected into each

Digitized by Google

left finger with the same psychic effect. On one occasion he remarked that the alcoholic injections seemed to release something in the left side of the head.

Time after time this soldier would report for treatment remarkedly improved and highly encouraged. At other times he was tremulous, fearful and deeply depressed. Trouble of some sort continued to cross his path. Always impressionable, the slightest annoyance invariably aggravated the disability and increased his nervousness. A chance meeting with his alleged unfaithful wife, a death in the hospital and the notification of his father's demise were some of the numerous perturbations which retarded his progress. As our observations proceeded, we finally were convinced that the Canadian medical officers made a proper interpretation of his mental state. He was not only poorly equipped to withstand ordinary strains and stress but wholly incapable of adapting himself to his environment. Many of his statements proved to be base prevarications, and he made claims that were extravagant and oftentimes sensational. His chief ambition was to pose as a wounded hero, and he habitually feasted on pity and sympathy. One day I saw him marching in a military parade, leaning heavily on a cane and carrying the left arm in an unnecessarily awkward position for the evident purpose of attracting attention. His expression of sadness and dejection was pronounced.

For a while the condition, though greatly improved, remained somewhat stationary, and we decided to take a firm stand with him. Instead of remaining at the hospital to follow instructions, he was wandering about the streets making acquaintances of a questionable character. The odor of liquor had been detected on his breath; but this he stoutly denied. He did, however, admit that persons had stopped him on the street and urged him to forsake medicine and seek relief at the hands of various cults whose cures, they declared, were more permanent.

We openly accused him of loafing on the job, and he was told that it looked very much as if he was not especially anxious to regain his health so long as he enjoyed monthly pensions in the amount of $62. This seemed to jar him, so to speak, and he instantly retorted that if he could be convinced that the paralysis would never recur, a full recovery would probably follow. He also admitted that his general sense of well-being was greatly improved; that the paralysis was ever so much less; that the nervousness and insomnia had practically subsided, but that he was constantly haunted by the fear that a piece of shrapnel had penetrated his brain, as the examining officers so often declared. Another interesting assertion was the confession that more than once he felt as if his health had been restored, but some excitement would arise to blast his hopes.

Digitized by Google

At the final examination, May 15, 1919, the patient looked well and was adequately nourished. He had gained 10 pounds in weight. He walked alertly with a slight limp, favoring the left leg. He was able to stand erect with the heels and toes together, and could stand on either foot unassisted. No clonus or Babinski toe response prevailed. He carried the left arm in an almost normal position, but there remained a moderate contraction of the thumb and first finger of the left hand which interfered with the fine movements of the left hand. The grasp of the left hand and the strength of the left arm and left leg were vigorous. The heart action was normal. The pulse rate was 88. The blood pressure measured 130 mm. systolic and 98 diastolic. The depressed flesh wound of the left scalp remained unchanged. The patient was able voluntarily to extend the great toe of each foot while the remaining toes retained their normal position. The tendon of each extensor hallucis longus was still prominent.

Mentally he was quiet and self composed. He had lost much of his emotionalism and apprehension, but was still morbidly impressionable. He was optimistic as to his recovery, which he believed was not far distant. He stated that during the excitement incident to the recent socialist uprising in the Cleveland streets, May 1, he almost forgot that he was a cripple.

COMMENT

The case is of interest in consequence of the confusing nature of the neurologic symptoms. Our initial examination disclosed a contracted left hand, a decided weakness and limp of the left leg, plus an apparently complete Babinski phenomenon of the left foot with a more or less pronounced clonus of the left ankle and patella. These clinical observations were also detected and recorded by the Canadian medical officers, and unquestionably influenced their opinion that the affliction was an organic hemiplegia and therefore a total disability.

At first we readily would have agreed with this classification; but the facts that the soldier sustained a slight flesh wound of the left scalp followed by paralysis of the same side of the body, including the face, and that subsequent roentgenograms of the skull and Wassermann tests of the blood and spinal fluid

Digitized by Google

were reported by competent observers to be negative aroused sufficient doubt in our minds to warrant delay in forming a definite conclusion.

After one month's observation and treatment we became convinced that the disability was none other than the expression of a hysterical disturbance. The early subsidence of the suspicious nerve signs and the marked general improvement by suggestive measures only, the abnormal impressionability of the individual, his inability to withstand the ordinary pin pricks of life, the pronounced emotionalism, and the eye manifestations did much to shape our diagnosis.

Little significance was placed on the clonus of the left ankle and patella, as this symptom frequently occurs in functional disorders and is therefore worthless as a pathognomonic sign.

The possibility of an admixture of hysteria and an organic lesion was carefully considered and eventually excluded. Nor did we overlook the importance of the reported observations of experienced neurologists that hysteria has never developed on the battlefield or when the soldier is still in danger, but only when he finds himself in relative safety. This patient is unable to give any reliable data as to when the paralysis first appeared. During the first twenty-four hours he was dazed and confused, but he entertains a vague impression that the condition developed while en route to the hospital in a military train.

The question now arises as to whether or not the alleged Babinski response of the left foot was genuine and therefore pathologic. We are inclined to doubt it and prefer to interpret the phenomenon as a psychogenic movement of resistance. On the other hand, when the sole of the left foot was gently stimulated there was a prompt, though slow, dorsal excursion of the large toe, and a simultaneous plantar flexion of the four external toes, symptomatic of the Babinski reaction. Perhaps the undue prominence of the tendon

Digitized by Google

of the extensor hallucis longus as well as the patient's ability to cause voluntarily a full dorsal movement of both large toes, while the other toes retained their normal position, may have influenced the behavior of this symptom.

In medicolegal work, instances are on record in which litigants have ingeniously succeeded, after some practice, in producing a full upward movement of one or both large toes, in much the same manner as certain persons have learned to wriggle an ear or wrinkle half of the forehead; but this patient emphatically denies that anything of the sort ever entered his mind.

The man has been under systematic treatment for a period of four months; and, while he is still partially disabled, a full recovery, we believe, may reasonably be anticipated.

Unfortunately, he has been subjected to innumerable examinations and tests; has listened repeatedly to discussions and controversies concerning his condition; and during his long residence in various special military hospitals, has come in intimate contact with all sorts of nervous invalids. All of this has undoubtedly tended to impress on his susceptible mentality the conviction that he is an incurable paralytic. It is also important to note that as a result of his extensive medical experience he has acquired a knowledge of "traumatic hemiplegia," which is interesting if not instructive. Then, again, if his disability has earned for him monthly pensions sufficient to satisfy his ordinary needs, without exertion on his part, there is little incentive to call into action his higher control. On the contrary, it is apt to operate, consciously or unconsciously, as a deterrent to his recovery.

Finally, it may be added that this clinical report serves the additional purpose of pointing out with what degree of perfection hysteria may simulate organic disease, and also emphasizes the difficulties one experiences in avoiding errors in diagnosis.

Digitized by Google

ABSTRACT OF DISCUSSION

Dr. Alfred Gordon, Philadelphia: The Babinski sign has been reported to have been found in cases of hysteria, but a careful examination will always reveal some error in the procedure or the method of bringing out the reflex. One has to stroke very gently the sole of the foot in order to bring out a genuine Babinski without dorsoflexion of the entire foot. With reference to the knee jerks in hysteria one must equally be careful in interpreting the reflex. A brusque, abrupt, sudden elevation of the leg is more in favor of a diagnosis of functional disease than of organic disease. Here again the case reports do not give a detailed account of the procedure employed or a detailed description of the phenomenon. With regard to the compensation question in functional nervous disease, there is a tendency at present to regard hysteria as malingering on the part of the patient. At the meetings of French neurologists the question was raised especially and for a special detailed discussion by the most eminent men. At first there was an inclination to decline compensation for cases of functional nervous disorders, hysteria included, and only one man, Pitres, of Bordeaux, raised his voice against such a decision, discussing at length the injustice rendered the individuals in this class. Another meeting was held for the same purpose and finally the entire association came around to Pitres' views, and 20 per cent. disability was considered as a standard for hysterical paralysis or other functional manifestations.

Dr. Tom A. Williams, Washington, D. C.: Two fallacies in such cases must be guarded against. One is mistaking for the extensor reflex the true Babinski, the defense reaction. Another is the simulation of the Babinski on the part of the patient. The defense reaction can readily be differentiated from the true Babinski phenomenon, if one knows the behavior of each. With reference to ankle clonus, it is not difficult for a patient to simulate this. The experienced neurologist, however, can make the differentiation, and if not an experienced neurologist, by a kymographic tracing. There is another interpretation in some of these cases, namely, that the patient has had a lesion capable of producing disorder of the pyramidal tract which will produce that sign and which rapidly disappears, such as meningeal irritation. The patient grafts the hysteria on the organic condition, and when he is examined one discovers only the hysteria and wrongly imagines the former reactions are due to the hysteria. The criterion of the case is the fact that the phenomenon can be induced and removed by persuasion and suggestion, in which case it cannot be an organic phenomenon.

Dr. Albert E. Sterne, Indianapolis: We have practically all had similar experiences, or will have like experiences at this time of returned soldiers, for reexamination is often

Digitized by Google

requested by the war risk department and a number of cases will present similar complexities which we are somewhat at a loss to interpret. We are laboring between two emotions, ourselves, in making our reports on these cases; namely, that we want to do justice to all the boys coming back who have been injured, but at the same time protect the government and ourselves from fraud. Whether organically injured or functionally disabled makes little difference, because a man functionally disabled is almost as badly off and sometimes worse off than one with a palpable bodily injury. The government compensations will amount in time to terrific figures and we are all anxious, naturally, to see that compensation is not awarded where it is not due. The suggestions grafted on these men in their prolonged convalescence in various camps must not be overlooked. I am convinced that the great majority of these boys do not desire to remain invalids; they are not in the malingerer class, but they exaggerate. There is a tremendous proclivity in the average normal individual to exaggerate symptoms, either through self-pity or the desire to excite sympathy. We must not be too sure about the revelations the reflexes present. Where the reflexes are seemingly positive, if tested and retested often enough they may be found changed. They are not always consistent. You should make the patient feel that you are with him, as much as possible, but are not going to encourage any unfitness on his part that can be cured, or encourage him in functional invalidism.

Dr. Lewis J. Pollock, Chicago: The important fact as to compensation for the war neuroses is not that the patients are not going to be compensated enough for their disability or that the government is going to be cheated out of money wrongly applied. The important thing is to get the boys well. If you hold out the promise of a compensation of a case of hysteria you will afford him an opportunity for continuing the functional condition. If it is decided that there is to be no compensation for functional disease that disease will disappear much more rapidly. The best compensation for a neurosis is its cure, and any other form of compensation is a sad commentary on our therapeutic efforts.

Dr. Hugh T. Patrick, Chicago: I understood Dr. Williams to say that if this were hysteria it would immediately disappear under persuasion or suggestion. If he would modify that to "immediately disappear under effective persuasion" I would agree with him. But the fact remains that some do not disappear under persuasion or suggestion. Hysteria in soldiers is more easily treated than hysteria of civil life and yet the records show that they have been subjected to persuasion and suggestion for many months at a time and have not been cured until they reached the man who made the suggestion which appealed to the patient and then they

Digitized by Google

got well. I think it should not go abroad as the sense of this section that hysteria will at once disappear under suggestion or persuasion. I would like also to support what Dr. Pollock said about these functional cases. They also can be compared to the same thing in civil life. If a man or woman has traumatic hysteria and sues for $20,000 it is well known that in the vast majority of cases after litigation is ended the hysteria disappears. A few days after the signing of the armistice the vast majority of the war neuroses had disappeared. Consequently, the giving of compensation to a purely hysterical disability is a great mistake. In the prewar period any one who had the opportunity to see the thousands of cases of traumatic hysteria in Germany and found practically none in France because the laws of the two countries were so different, would come to the same conclusion.

Dr. W. S. Lindsay, Topeka, Kan.: I understood Dr. Williams to say that he did not consider that this case was organic. I have known of a case of cerebral tumor attended by convulsions where it was possible for the attendant to avert the convulsive seizures by engaging the attention of the patient. We cannot exclude cerebral disease because we must take into consideration the fact of cerebral inhibition.

Dr. Foster Kennedy, New York: The question of compensation of discharged soldiers suffering from hysteria is a matter of most potent interest at present and we should get our minds clear on this question. The British government pensioned about 25,000 soldiers for what was called shell shock up to May, 1918. This involved a very large sum of money and a large number of men. Those cases furnished the medical view of what shell shock is. The doctors were wrong and the soldiers were right. The soldiers knew it was a neurosis associated with the desire to preserve life, also a breakdown of the man's adaptation to environment. It was not the result of petechial hemorrhages in the brain. The medical men advised the government and the result was this method of compensation. I understand the French take the position that they will not compensate for a functional condition. That seems to be a serious thing for the individual and in these days of the civilian army the individual should be given a certain amount of consideration. It is not true that you will cure your hysteria patients if you do not compensate them. You will have to decide what is hysteria and what is organic disease first. If you can be sure of your diagnosis then you can decide accurately. As a matter of fact, most people when they get an injury of the arm in war go through a period of defense immobilization. If they are told that the arm is paralyzed then the seed of hysterical paralysis is laid. You must make it a rule to be very sure of your diagnosis, or you must have a very strong compen-

Digitized by Google

sation board to decide what is hysteria in some cases and what is organic in others because the chief error that is being made all the time is a question of diagnosis.

Dr. Edward E. Mayer, Pittsburgh: I do not believe it wise to assume the same attitude toward war hysteria now as was done before the armistice. I agree with Dr. Patrick and Dr. Pollock that the feature of noncompensation was advisable during the war and that it would have been better had all the armies practiced it from the beginning. But there is a different angle to the problem now. Though the fear of the battlefield has been removed and the instinct of self-preservation is no longer disturbed, nevertheless continued disability not only implies a fixation of symptoms but a protective mechanism which needs removal and which is in most cases due to other factors than the war situations and experiences. They are unable to care for themselves and must be given hospital facilities, vocational work and competent neurologic oversight until recovered and after-care help so that no economic worry affect them after discharge. The War Risk Insurance Bureau, I know, realizes this problem but it is complicated I find by the fact that many of the war neuroses have been discharged and must themselves take up with the War Risk Board their problems instead of its having been done by army neurologists in advance of discharge. Let us, therefore, separate the compensation problems of the war and the compensation problems of after-war. In the latter case, compensation is deserved and necessary and we neurologists must above all recognize it.

Dr. Julius Grinker, Chicago: At the meeting of another section I heard some one remark that the diagnosis of hysteria is only a sign of the examiner's perplexity. I did not think I would hear a similar expression voiced in this section. Many people mean different kinds of disease when they speak of hysteria. In hysteria there is simulation, but it is of the unconscious kind. This we know to be only one of the many symptoms of hysteria. I think the speakers who preceded me had in mind conscious as much as unconscious simulation when they spoke of war hysteria. After everything has been said on hysteria one thing is certain, namely, that it exists and is a functional disease and must be treated as such. Not all cases of traumatic hysteria which have been awarded compensation in industrial life have thereby been cured because the disease is not synonymous with simulation. No one remedy will cure all forms of hysteria. Whatever name you give the disease, it exists, and as medical men we are justified in recommending compensation to those who become genuinely hysterical as the result of trauma whether in peace or war.

Dr. Harry H. Drysdale, Cleveland: Dr. Williams is inclined to regard the curability of hysteria a rather easy

Digitized by Google

matter, but it has not always been so with us, especially in the cases of long duration. The case I have brought to your attention was complicated by the award of a liberal pension which consciously or unconsciously has served to deter recovery. The differentiation between simulation and true hysteria should not be difficult if the investigation is carefully conducted. When necessary, suitable observation will clear up any doubtful points. I am heartily in accord with the remarks of Dr. Pollock and Dr. Patrick.

Digitized by Google

CONGENITAL TUMOR OF THE BRAIN (TELANGIECTASIS) AND ASSOCIATED CEREBRAL MOVEMENTS

WILLIAM G. SPILLER, M.D.
Professor of Neurology, University of Pennsylvania
PHILADELPHIA

The occurrence of congenital telangiectasis of the brain, diagnosed clinically and treated by operation is infrequent, and for this reason I have made a study of the case here recorded. It affords the opportunity to discuss associated movements of the limbs resulting from cortical defect.

REPORT OF CASE

History.—J. C., aged 12, was admitted to the University Hospital, June 17, 1918. The mother of the boy was exposed to roentgen rays when three and one-half months pregnant with this boy. Another child was held on the lap of the mother while it was being roentgen rayed. The birth of the patient was normal. When he was 2 or 3 months old his right upper limb was observed to be paralyzed, but it probably had been so from birth. The mother noticed when the child was about 1 year old that the right lower limb was paralyzed, because the child made no effort to crawl or stand. Since his birth he has had a telangiectasis of his forehead over the left eye, one in the left side of the scalp near the front hairline, a similar one in the left eyeball, and a small one on his back.

The first convulsion occurred May 1, 1918. This implicated the whole body and was with unconsciousness. The second attack occurred four weeks after this one and was general, and similar convulsions occurred later. He learned slowly at school and was backward in his classes.

Examination.—He wrinkles his forehead slightly. He closes both eyes well together, but cannot close either separately. In showing his teeth some weakness of the right side of the face is detected. The tongue is protruded awkwardly. He cannot close the right hand alone at all, but when he closes the left hand there is associated movement of the same kind with the right hand which the boy cannot prevent. Flexion at the left wrist and often even at the elbow produces a similar movement of the corresponding part of the right upper limb. The voluntary movement of the right upper limb at the elbow and shoulder is impaired. The movement at the right wrist is lost except in

Digitized by Google

association with movement at the left wrist. The right hand is flaccid. The dynamometer registers 2 degrees in the right hand, 10 degrees in the left hand. The right upper limb is poorly developed and the right hand is much smaller than the left. Diadokokinesis is normal in the left hand and the test does not produce associated movements in the right hand. Stereognostic perception is lost in the right hand and the boy can tell only whether the object is cold or warm with this hand. It is normal in the left hand.

He has imperfect movement in extension of the right lower limb and the power is not great. He cannot dorsally flex the right foot. Associated movements are present in the right toes. He cannot move the right toes alone, but when he moves the left toes the right toes move similarly, but associated movements are more pronounced in the right hand than in the right foot. He walks without difficulty.

Achilles and patellar tendon reflexes are exaggerated on the right side, normal on the left side. The Babinski reflex is doubtfully present on the right side, absent on the left. Temperature, touch and pain sensations are normal.

Operation.—Dr. Frazier operated on the patient June 26, 1918. A flap was made over the Rolandic fissure. On reflecting this, a dark-bluish discoloration with bulging of the brain appeared in the anterior and superior third of the opening, as though cavernous spaces of blood were beneath the dura at these places. The branches of the meningeal vessels were three or four times as large as they usually are and bled freely from the time the osteoplastic flap was reflected, and closure was attended with much difficulty. On incising the dura along the superior margin of the opening a tremendous venous channel was penetrated in which the blood welled until hemostatic clamps were applied. So much blood had been lost from various causes that no attempt was made to explore further.

Results.—July 24, 1918: The father reported that no convulsion had occurred since the operation. The boy has no increase of the right-sided weakness. He had a number of convulsions on July 29, but only one attack from this date to September 9.

April 11, 1919: His mother stated that his last convulsion had occurred in October, 1918, and was very slight. He had had no attack of any kind since and had been perfectly well, and had done well in school. Whereas formerly he had obtained very poor marks and had been observed to be deteriorating mentally, now he was bright and had recently obtained an almost perfect grade.

CASES MENTIONED IN THE LITERATURE

Angiomas have been classed by some as congenital tumors. The first case reported by Oppenheim in his paper on congenital brain tumor is the most interesting of his series. The patient had left hemianopsia since birth or early childhood. Sight had been absent in the left fields as long as he and his mother could remember; as a child

Digitized by Google

he had not seen food or toys placed on his left side, and yet he seems to have had no other signs of cerebral disease until he was 18 years old. At that age he had severe headache, paresthesia, paralysis of the entire left side, etc. These symptoms disappeared leaving some weakness of the left side. When 25 years old he had a second attack consisting of severe headache and vomiting and lasting about 14 days. At 26 years he had a third attack similar to the first and became paralyzed on the left side. The diagnosis of a lesion of the right optic thalamus and the adjoining region was made. It was supposed to be a congenital tumor which caused focal symptoms at different periods because of transitory swelling or hemorrhage, probably an angioma cavernosum.

In three out of Oppenheim's four cases the symptoms began in adult life, in one at 18 years, in another at about 23 or 24 years, and in the third at about 33 years. The relative late onset of symptoms in angioma is striking, also the possibility of repeated hemorrhage in such a tumor, but all of his cases seem to have been without either operation or necropsy. An angioma of the face was found in two of the four cases, and Oppenheim mentions that experience has shown such a lesion is frequently associated with angioma of the cerebral meninges, and he refers to Kalischer, Emanuel, Bruns and himself in this connection.

He mentions as congenital tumors of the brain dermoid cysts, teratoma of the hypophysis, angioma, possibly cholesteatoma and neuroma of the brain nerves.

These congenital tumors may cause symptoms in early childhood, and then be latent many years, or produce symptoms with long intermissions, or the symptoms may first appear in later life.

Oppenheim had only twice been able by biopsy to confirm the clinical diagnosis of angioma of the brain, although he had observed angioma a few times at operation in cases in which he had made the general diagnosis of brain tumor. He does not distinguish sharply between angioma and telangiectasis.

In the first of the two cases reported by Ernest Sachs there were fever and convulsions confined to the left side of the body beginning three months after a fall. The possibility of hemorrhage from the telangiectasis is suggested by this, although Sachs does not accept this

Digitized by Google

as a factor. A left-sided paralysis developed also and disappeared in a month. Then the patient was well about one year and because of the reappearance of jacksonian convulsions an operation on the brain was performed. A huge bluish mass of vessels protruded when the dura was opened, and was so dense in places that the underlying cortex could not be seen. An attempt to remove this mass at a second operation resulted fatally.

The second case was that of a patient with a pale telangiectasis over the forehead. An angiomatous process was found in the dura, which on reflecting the dura was found to have numerous connections with the pial vessels. All the vessels connecting with the pial vessels were ligated and the dural mass was ligated above and below. Two days after the operation the patient had a violent convulsion, but since then had been well. It would seem as though this extensive ligation of the vessels had not had any permanent serious result.

Sachs found very few cases in the literature like his second case. He mentions that the process in all three of Cushing's cases was in the dura, and that Cushing does not mention the possibility that an identical process might occur on the cortex.

Sachs disputes the propriety of calling these tumors angiomas and quotes Virchow and Adami in support of this opinion. He emphasizes that a telangiectasis is a congenital dilatation of capillaries without any new-formed blood vessels, while an angioma is a new growth and only properly so-called when new blood vessels are formed. These distinctions are lost sight of in the literature. He also refers to the existence of telangiectasis elsewhere on the body, especially the face, and mentions that this was true of Pean's case and of Cushing's.

In the case reported by Castex and Bolo the lesion was designated by them as angioma venosum racemosum and extensive ligation of the vessels was followed by recovery. No writer so far as I know has spoken of the danger of extensive ligation, and yet I have seen serious symptoms result in two cases from this procedure. In one case a hemorrhage occurred into the brain, and in the other hemiplegia resulted. I would therefore suggest caution in tying many vessels at one time, in cortical telangiectasis.

In my patient the numerous angiomas of the surface in connection with symptoms of cortical lesion indicated that the latter probably also

Digitized by Google

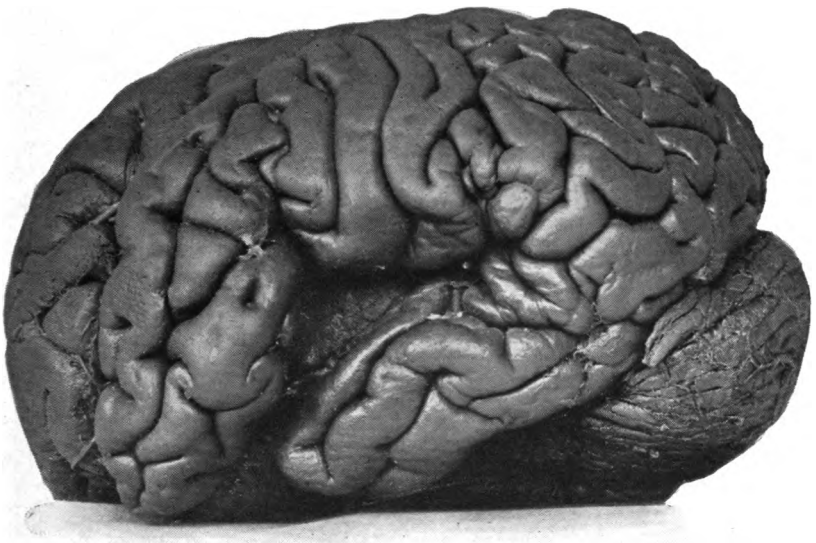

Fig. 1.—Porencephaly of the left cerebral hemisphere.

Digitized by Google

Fig. 2.—Another view of porencephaly of the left cerebral hemisphere.

Digitized by Google

Fig. 3.—The anterior part of the medulla oblongata from the case of porencephaly. The left anterior pyramid is wanting and the right anterior pyramid is unusually large. The left fillet is much smaller than the right.

Digitized by Google

Digitized by Google

was of the same vascular character, and I advised operation with this probability in mind. The operation revealed extensive telangiectasis of the motor cortex. Caution was exercised by Dr. Frazier to avoid ligating too many vessels, and no complications occurred. I have observed two cases of teratoma of the region of the medulla oblongata both with successful operation — one by Frazier and one by Elsberg.

The associated movements of the right side, the paralyzed side, were very marked in the case I have described. Such movements are well known and are most pronounced when the paralysis has dated from birth. Very rarely associated movements occur in people otherwise normal, who are unable to use either hand without a corresponding movement of the other hand, and cases of this character are attributed to Damsch, Fragstein, and Brissaud and Sicard by Lewandowsky. I have seen two striking examples of this: one case was presented by Drs. C. W. Burr and C. B. Crow before the Philadelphia Neurological Society; the other was exhibited at the last International Medical Congress held in London, but I have never seen any report of this case published.

The case presented by Burr and Crow was as follows: The patient, a man, had had the condition since infancy. Whenever he made any complicated voluntary movements with the fingers or wrist of either hand the other hand involuntarily made the same movements. Very simple motions such as flexing or extending one finger could be made independently, but even then, as a rule, there was a similar simultaneous movement of the corresponding finger of the other hand. The condition was confined to the hand muscles and did not appear when the upper arm alone was used. The feet and toes were not affected. By strict attention he could prevent the unused hand from moving, but he could not, for any length of time, keep his attention fixed on what he was doing and on inhibiting the hand. He did his work as a seaman well until he was ordered to climb a rope. He demurred, and it was found that on letting go with the lower hand to reach for a higher grip on the rope he let go with the upper hand and fell.

In discussing this case, I suggested that each hand of this man was innervated from both sides of the brain equally or both were innervated from one side of the brain alone.

Digitized by Google

AUTHOR'S OBSERVATIONS

The central nervous systems from two cases in my possession throw light on the mechanism of these movements. Unfortunately, the clinical history is very deficient in each case, and I do not know whether associated movements were present or not. Both established the fact that the anterior pyramid on the side opposite to the lesion may be unusually large when the other anterior pyramid is absent from a congenital defect of the pyramidal tract of one side.

One was a case of *porencephaly*. The brain was from a low-grade boy, whose memory was fairly good, who sang, but never learned to read. He was partly paralyzed on the right side and never used his right hand. An opening extended into the left lateral ventricle, and the walls of this ventricle were continuous with the external surface of the brain (Fig. 1), and the base of the left frontal lobe presented a condition of agenesia. The anterior pyramid on the left side was absent (Fig. 3) so that no motor fibers could have passed into the cord from the left cerebral hemisphere through the anterior pyramid. The right anterior pyramid was unusually large. The fillet on the left side also was much smaller than that on the right side. Inasmuch as he was reported to have been only partially paralyzed on his right side, whatever limited voluntary power he had on this side must have come from the right cerebral hemisphere.

The second case was that of a man (G. S.) aged 66, who entered my service at the Philadelphia General Hospital, Jan. 24, 1916, a few days before I went off duty, and was never studied clinically by me.

He had left-sided palsy said to have dated from birth, but this had not prevented him from working. He was able to go about, but had very little use of the upper limb, and could not use the left hand.

Shortly before Christmas, 1915, he seems to have had a stroke, and he lost power in both lower limbs. His mentality was much impaired. The grip in the right hand was good.

A note was made Feb. 25, 1916, that he had partial paralysis of his upper limb and of both lower limbs, and was slowly regaining strength in the lower limbs. He died Aug. 2, 1916. A small area of softening was found in the upper part of the left lenticular nucleus and above it and was probably the cause of the attack in 1915. The lateral ventricles were dilated, but the right one was very much more so than

Digitized by Google

the left. The right anterior pyramid was exceedingly small, while the left was abnormally large. The right lemniscus was also much smaller than the left.

The cervical region, on the whole, showed diminution in size of the left anterior horn and of the number of cells it contained. There were sections in which this distinction was not so evident. The left lateral column was considerably smaller than the right in the cervical region, but showed no signs of overgrowth of neuroglia. In the lumbar region it was impossible to make any such distinctions between the two sides of the cord. There was, therefore, a moderate hypoplasia of the left side of the cord in the cervical region.

In both these cases there was partial hemiplegia dating from birth, and in both the anterior pyramid on the side opposite to the lesion was unusually large, while the other anterior pyramid was entirely absent in the case of porencephaly and exceedingly small in the case of hydrocephalus. It is reasonable to conclude that the intact pyramid contained homolateral motor fibers, and in this way some movement was still possible in the hemiplegic side. In 1895, while working in Dejerine's laboratory I made a study of the spinal cords in many cases of hemiplegia in which death followed the lesion in a comparatively short time—that is, in a period permitting the use of the Marchi method of staining—and was able to demonstrate degenerated fibers in both crossed pyramidal tracts, more in the side opposite to the lesion, in many of the cases, from an unilateral lesion of the cerebrum. This was evidence that the degenerated fibers came from the side of the brain in which was the lesion and indicated that each side of the brain supplied both crossed pyramidal tracts of the spinal cord, though in unequal degree.

COMMENT

It is interesting to note that the boy with telangiectasis of the cortex and pronounced associated movements developed convulsions which were not jacksonian but implicated both sides of the body. Probably these convulsions, like the voluntary and associated movements, originated in the right motor cortex. The loss of stereognostic perception of the right hand might be attributed to the lesion of the left parietal lobe, and it is interesting to recall that the lemniscus on the side of the cerebral lesion in the two cases with necropsy was very much

Digitized by Google

smaller, as though the decrease in size might have been a form of retrograde degeneration or possibly a hypoplasia. I have seen a diminution in size of the lemniscus in several cases of lesion of the cerebrum.

The diminution of the whole right side of the body and limbs of this boy probably was in association with defect of the motor tract from the left cerebral hemisphere. The case of hydrocephalus showed some diminution in the size of the motor tract in the spinal cord from the right side of the brain. It is common to find arrest of muscular development in the paralyzed side in early acquired or congenital hemiplegia, and probably this is dependent on defect of the motor supply of the corresponding side. At birth the muscles may be developed independently of the motor nervous system, as von Leonowa first showed, but at a very early period of extra-uterine life—exactly at what period is not known—the motor nervous system takes control of the muscular system, and defect of the former leads to defect of the latter.

BIBLIOGRAPHY

Oppenheim: Neurolog. Centralbl. **32**:3, 1913.
Sachs: Am. J. M. Sc. **150**:565 (Oct.) 1915.
Castex and Bolo: Deutsch. Ztschr. f. Nervenh. **52**:356, 1914.
Damsch: Ztschr. f. klin. Med., Suppl. Vol., 1891, p. 170.
Burr and Crow: J. Nerv. & Ment. Dis. **40**:300 (May) 1913.
Spiller: Ibid, **40**:399 (June) 1913.
von Leonowa: Neurol. Centralbl. 1893, p. 218.

DISCUSSION

Dr. H. H. Hoppe, Cincinnati: In a large experience I have seen but one case of telangiectasis of the cortex. This occurred in a boy previously well up to the age of 19, when he developed attacks of jacksonian epilepsy. The most careful examination carried on over a period of three to four weeks failed to reveal any signs of disease of the brain as manifested through physical signs. There was no weakness of the right side of the body, and not even the slightest indication of the Babinski sign of the sole of the right foot. On account of the fact that the boy had jacksonian seizures, it was deemed advisable to lay open the cortex and this condition of telangiectasis was found. In the majority of cases that I have read of there is no evidence to show before operation what is going to be found in the cortex. In the second place, I wish to question seriously after you have found this condition of telangiectasis whether it is wise to do anything further or not. The danger of ligating the vessels which supply the cortex of the brain has to be considered. In the case I referred to, the boy was made distinctly worse by the operation. He developed

Digitized by Google

a hemiplegia which prevented his earning his living, which he had been able to do before, and the operation did not improve his epileptic seizures. In fact, six months afterward they returned, and were just as bad if not worse than they had been before. While we may be justified in laying open the cortex to establish the diagnosis under these conditions, I doubt whether it is wise to tie off the arteries.

Dr. William G. Spiller, Philadelphia: I cannot agree with all that Dr. Hoppe has said. I think it is better to tie a few arteries at the time of operation and the rest later. That was done in my case and the patient was greatly improved. This has been found to be a successful procedure. Whether it will remain so or not I do not know. In many cases you cannot diagnose angioma of the cortex, but if you find similar growths on the head or face you may conclude the lesion in the brain is angioma.

Digitized by Google

A CONTRIBUTION TO THE HISTOPATHOLOGY OF EPIDEMIC ("LETHARGIC") ENCEPHALITIS *

PETER BASSOE, M.D.
Associate Professor of Nervous and Mental Diseases, Rush Medical College

AND

GEORGE B. HASSIN, M.D.
Attending Neurologist, Cook County Hospital

CHICAGO

The various symptoms of epidemic ("lethargic") encephalitis—somnolence, stupor with or without slight elevation of temperature, together with ocular and other cranial nerve paralyses, etc., may occur in many cerebral disorders. We may mention the hemorrhagic superior polioencephalitis of Wernicke, the cerebral form of poliomyelitis, the sleeping sickness caused by trypanosomiasis, syphilitic meningitis, paretic dementia, encephalitis caused by anthrax (Fulci[1]) or other infectious diseases, as tuberculosis, cerebrospinal meningitis and especially influenza. Here also belong cases of poisoning with carbon monoxid, sulphuric acid (Wernicke[2]) and meat and fish (botulism). Any of the types of encephalitis mentioned may so resemble the clinical picture and course of the epidemic ("lethargic") form that they, undoubtedly, have been and are confounded with the latter. Especially striking is the clinical similarity between lethargic encephalitis and the sleeping sickness caused in man by the *Trypanosoma gambiense*, hence "lethargic" encephalitis is popularly called "sleeping sickness." In addition, from our histopathologic studies of three cases of so-called lethargic encephalitis we are forced to the conclusion that the latter, though a disease *sui generis, does not differ histologically from the African sleeping sickness due to trypanosome infection.*

* From the laboratories of pathology of Cook County and Psychopathic Hospitals, Chicago.

1. Fulci, F.: Die acute hämorrhagische Leptomeningo-enzephalitis bei der Milzbrandinfektion des Menschen, Nissl-Alzheimer's Arbeiten 6:161, 1913.

2. Wernicke, C.: Lehrbuch der Gehirnkrankheiten 2:229, 1881, Berlin.

Digitized by Google

Of the three cases here described, two have been briefly reported by one of us (Bassoe[3]) and a third case, in a baby 4 weeks old, came under observation of Drs. C. G. Grulee and Peter Bassoe not long ago.

REPORT OF THE THIRD CASE

History.—A female child, 4 weeks old, vomited frequently for two days, and then repeated attacks of contractions of the diaphragm set in. When admitted to the Presbyterian Hospital, March 14, 1919, she was stuporous, with temperature ranging from 98 to 102 F. and a leukocyte count of 9,950. A laparotomy was performed and appendicostomy made. The following day the temperature rose to 103.2 F. and subsequently generally ranged between 100 and 102. A few days later nystagmus and strabismus were observed. The child died on March 25.

Necropsy.—This was held by Dr. B. O. Raulston and the following anatomic diagnosis was made: moderate hyperemia and edema of the brain; hyperemia of lungs; distention of urinary bladder; bilateral hydronephrosis; moderate dilatation of ureters; recent unhealed laparotomy wound; open stump of appendix; fatty changes in liver; patent ductus arteriosus and foramen ovale.

MACROSCOPIC EXAMINATION OF CASE 3

Macroscopically, the brain appeared practically normal. The pia was somewhat opaque, especially around the cerebellum; it was congested, rusty looking, but easily detachable from the brain substance. The convolutions were flattened, the sulci not gaping, the ventricles slightly dilated, the gray matter distinct from the white substance. There were no hemorrhages, no signs of ependymal changes.

MICROSCOPIC EXAMINATION OF THE THREE CASES

The brains from these three cases and the spinal cord of the third case were delivered to the laboratory fixed in a 10 per cent. formalin solution. Portions from every region of the hemispheres and brain stem, as well as from the spinal cord, were placed in 96 per cent. alcohol (for celloidin sections); some were prepared for paraffin, and a great number of frozen sections were also made from almost every portion of the central nervous system. The sections were stained by various methods, such as Mallory-Jacob, Alzheimer-Mann, Herxheimer (scarlet red), Bielschowsky, Weigert's elastic stain, toluidin blue, thionin, hematoxylin-eosin and many others.

3. Bassoe, Peter: Epidemic Encephalitis, J. A. M. A. 72:971 (April 5) 1919.

Digitized by Google

The pia appeared infiltrated and frequently showed distended meshes containing congested, hyperemic vessels. The infiltration cells were mainly lymphocytes and plasma cells, about equally represented and especially numerous around the vessels. Numerous scattered cells were also seen in the meshes of the pial connective tissues (Fig. 1). Both the lymphocytes and the plasma cells appeared very densely stained, the latter showing a distinct cytoplasm and an excentrically located nucleus which exhibited the typical wheel-like arrangement of its chromatin and a distinct membrane. In contrast to these cells, the lymphocytes were represented by darkly stained, often homogeneous, nuclei, without any traces of cytoplasm. The latter was also absent in many plasma cells which on superficial examination resembled lymphocytes. The plasma cells were not always of a typical appearance, but they were very numerous. Besides the lymphocytes and plasma cells, there were other forms present, but much less numerous, namely, fibroblasts, polyblasts, macrophages and rod cells — *Stäbchenzellen* (Fig. 2). The fibroblasts usually appeared as large, oval or round nuclei, filled with numerous small particles of chromatin, surrounded by a well-formed membrane, but without any visible cytoplasm. They could easily be distinguished from the plasma cells and lymphocytes by their larger size and pale nuclei. Still fewer in number were certain other cells also with pale, irregular, often horseshoe-shaped nuclei, enclosed within a pale cytoplasm. These cells—polyblasts—were scattered singly or mixed with the three forms mentioned above.

The macrophages were exceedingly rare, often contained small particles of blood pigment, or distinct vacuoles, and an excentrically located nucleus. The macrophages were seen only in those places where blood pigment was present, mostly as very minute particles.

Toluidin-blue specimens frequently revealed a great number of rod cells (*Stäbchenzellen*) especially near the vessels. They exhibited an oblong, distinctly stained nucleus, with chromatin dots, and very pale cytoplasmic processes, emanating from the opposite poles. Any other cellular elements, such as mast cells, gitter cells and polymorphonuclear cells were not found. The infiltration was in evidence all over the pia, that is, around every part of the brain, but was especially marked around the cerebellum, more so at the base, and it frequently involved the adjacent parenchyma. Figure 1 distinctly shows the invasion by the infiltration cells of the molecular layer of the cerebellum.

Digitized by Google

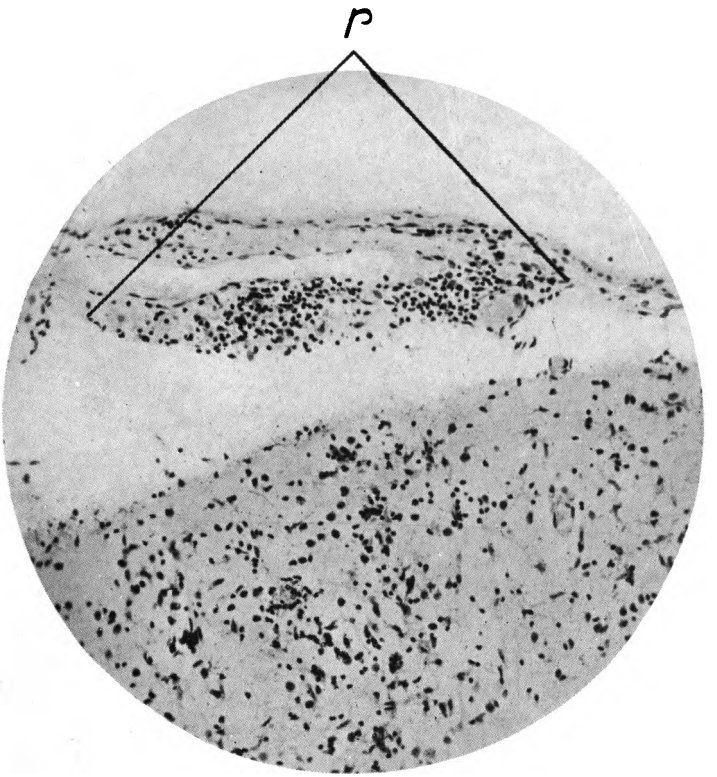

Fig. 1.—Infiltration of pia and cerebellar cortex. *P*, pia infiltrated and separated from the cerebellum. The molecular layer of the latter is much infiltrated by various cells as explained in the text. (Toluidin-blue stain, × 150.)

Digitized by Google

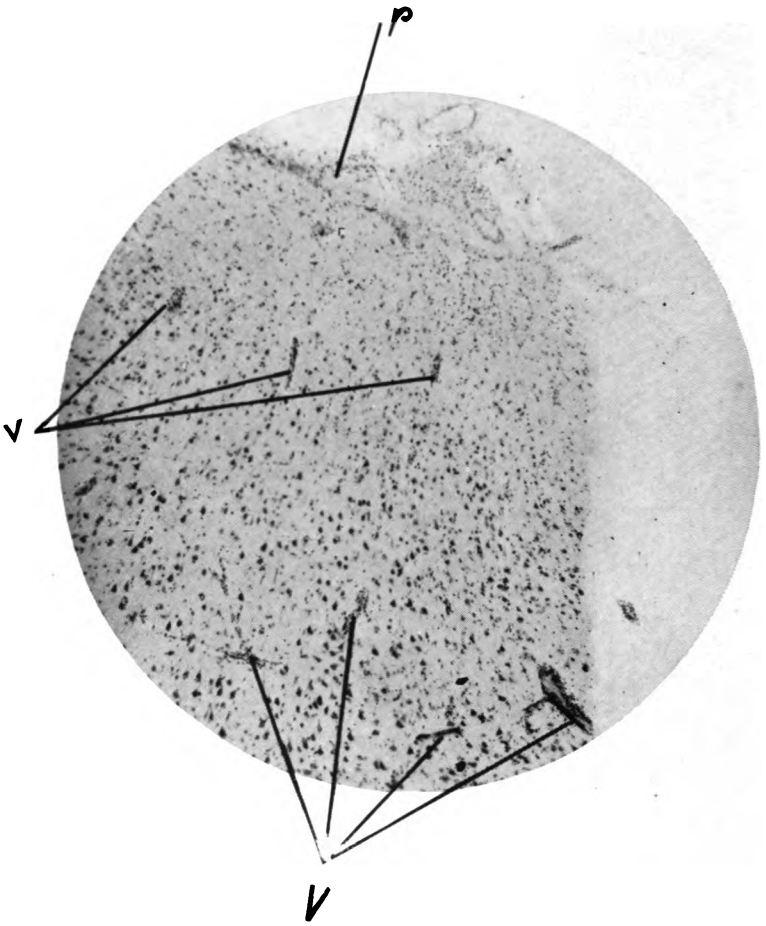

Fig. 3.—Cortex from the Island of Reil; the vascular infiltration especially in the deeper strata is very marked; the pia (*P*) is also infiltrated, but slightly. *V*, vessels. (Toluidin-blue stain, × 50.)

Digitized by Google

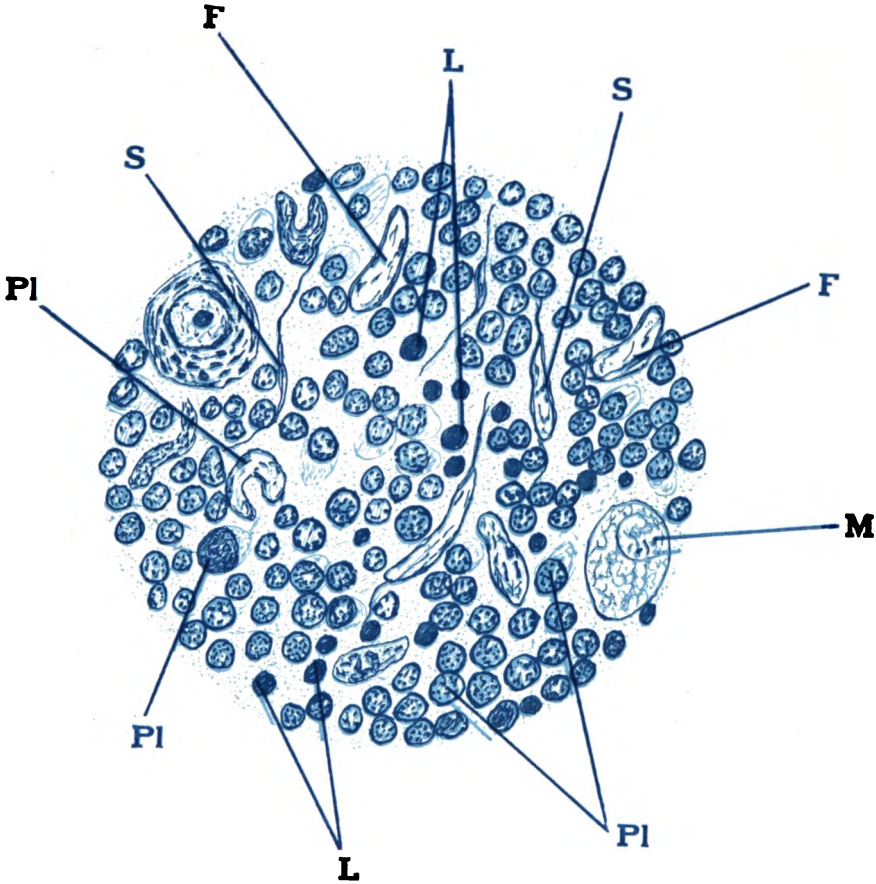

Fig. 2.—Types of various infiltration cells: L, lymphocytes; Pl, plasma cells; M, macrophage; Pb, polyblast; F, fibroblasts; S, *Stabchenzellen*. Toluidin-blue stain.

Digitized by Google

Digitized by Google

Fig. 4.—Pial vessels. The elastic membrane in the form of solid rings is very clear. (Resorcin-fuchsin stain, × 60.)

Digitized by Google

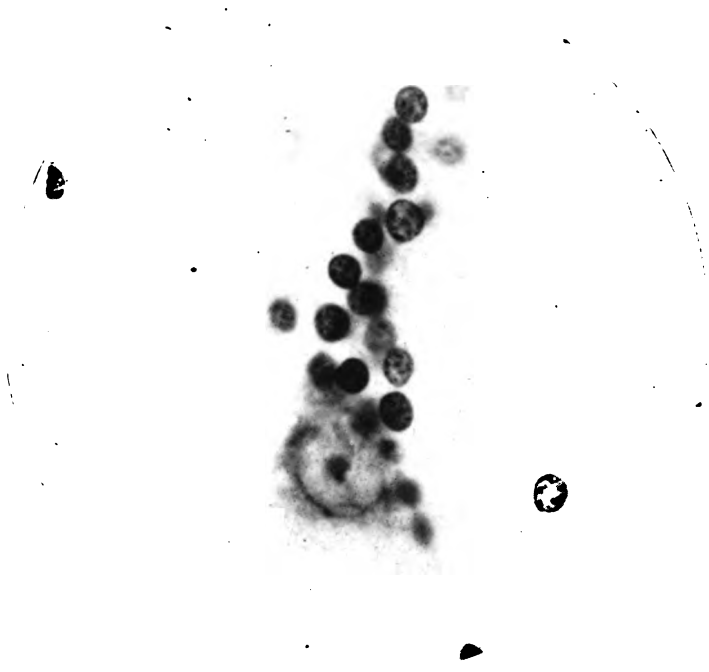

Fig. 6.—Type of neurophagia in the cortical and subcortical regions. The apical process as well as the right basal one are denesly covered by glia cells. The apical process is still visible beneath the glia cells. The cell body is very pale, the chromatin accumulated as a perinuclear cap. The round, free bodies scattered over the picture are also glia cells. (Toluidin-blue stain, × 100.)

Digitized by Google

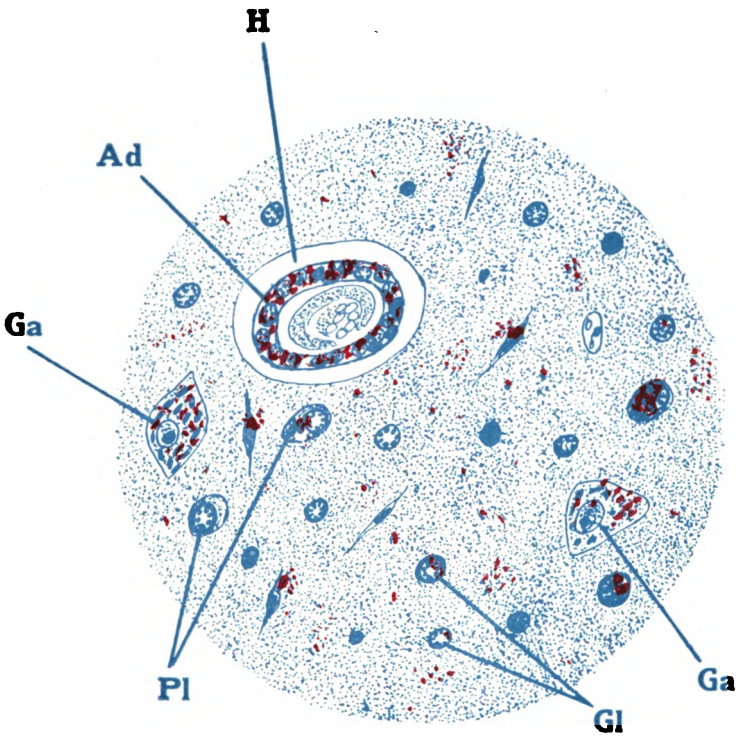

Fig. 5.—Substantia nigra. The ganglion cells, the glia nuclei, the adventitial space show fat globules which are also scattered all over the glia network in the form of minute droplets (Hematoxylin and Scarlet-red stain of Herxheimer.) *Gl*, glia nuclei; *Pl*, plasma cells; *Ga*, ganglion cells; *H*, His' space (Nissl's *Schrumpfraum*); *Ad*, adventitial space of Virchow-Robin.

Digitized by Google

Digitized by Google

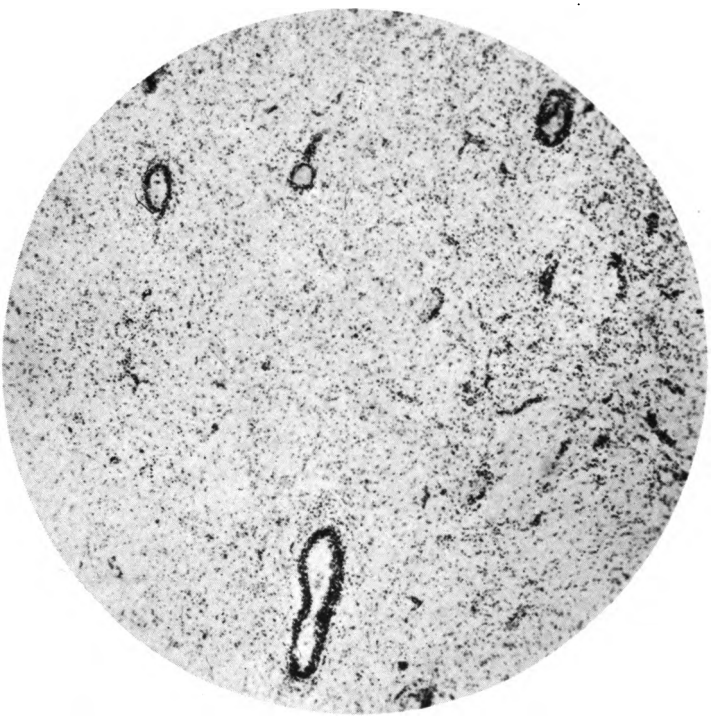

Fig. 7.—Infiltration of the optic thalamus, with numerous elements, explained in text. As compared with Fig. 5 the thalamus seems to be even more affected than the substantia nigra. (Toluidin-blue stain, × 60.)

Digitized by Google

Fig. 8.—Substantia nigra is covered by densely infiltrated vessels and numerous glia nuclei, especially to the right of the larger vessel. Some of the dots are free plasma cells and other bodies, as explained in text. (Toluidin-blue stain, × 110.)

Digitized by Google

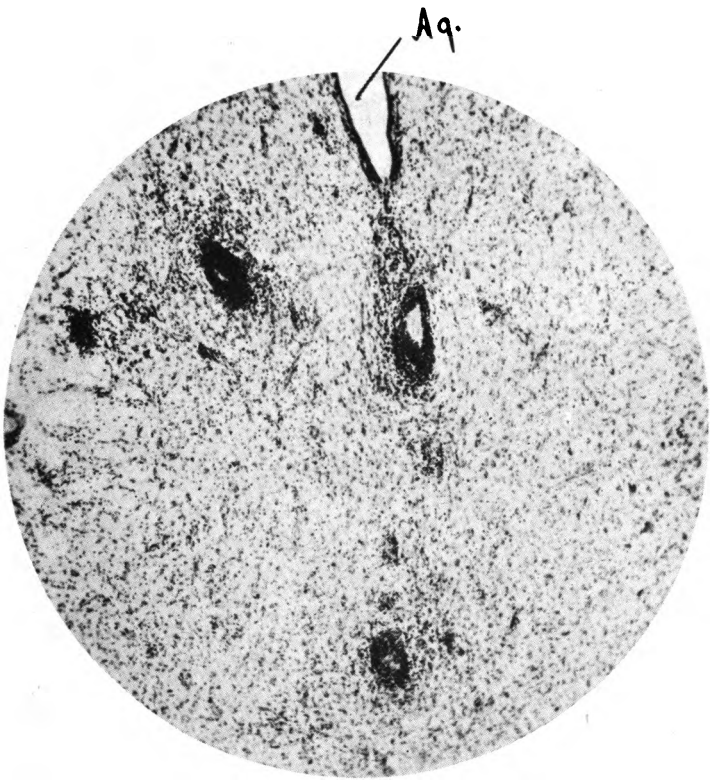

Fig. 9.—Tegmentum of pons. Infiltration of the vessels and of the formatio reticularis. *Aq,* Aqueducts Sylvii covered with ependyma cells. (Toluidin-blue stain, × 65.)

Digitized by Google

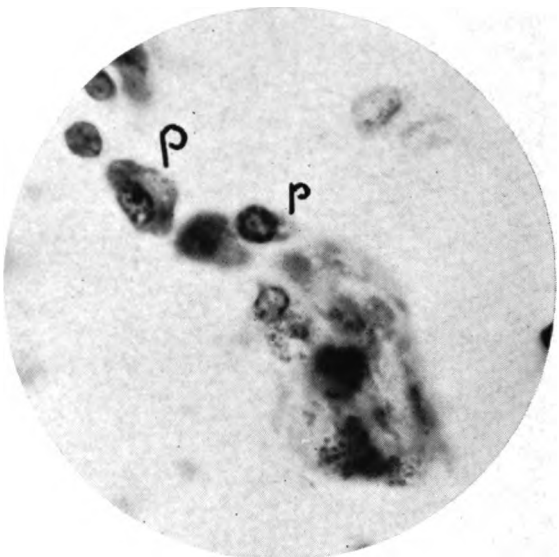

Fig. 10.—Typical plasma cells *(P)* along a dendrite of a deeply changed ganglion cell. (Toluidin-blue stain, × 1,200.)

Digitized by Google

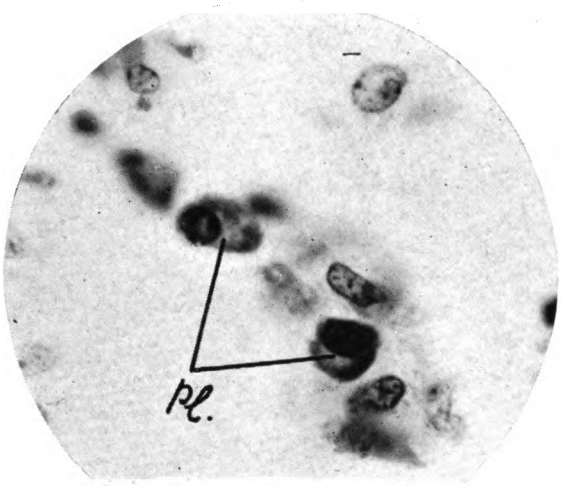

Fig. 11.—A degenerated ganglion cell (from substantia nigra) *invaded* and surrounded by plasma cells (*Pl*). (Toluidin-blue stain, × 1,200.)

Digitized by Google

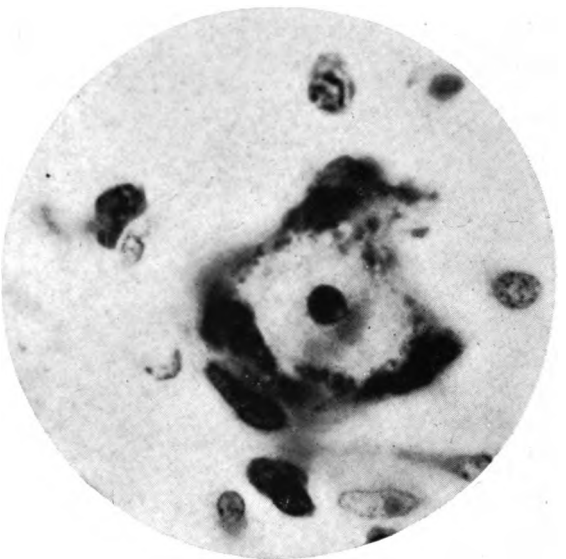

Fig. 12.—A destroyed ganglion cell from the subthalamic region surrounded by plasma cells. (Toluidin-blue stain, × 1,200.)

Digitized by Google

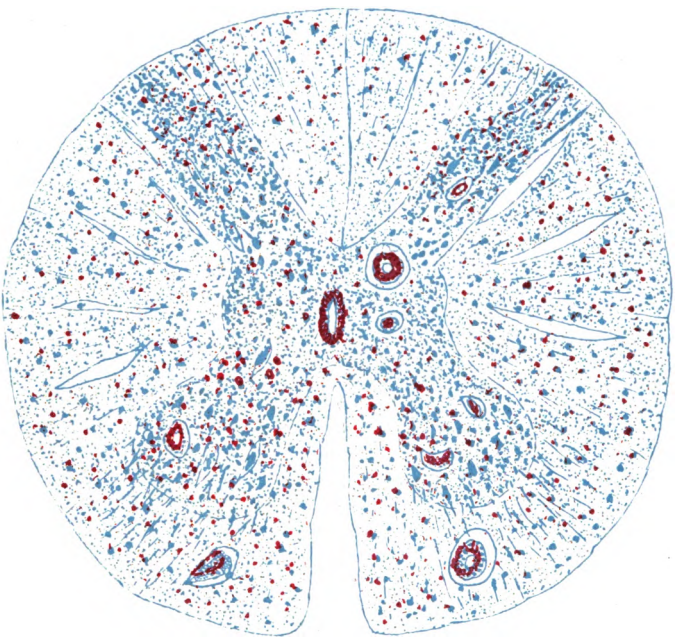

Fig. 14.—Spinal cord from Case 3 (lumbar region). The ependyma of the central canal, the blood vessels, the gray and white substances show an abundance of red spherules described in the text. (Frozen section; Alzheimer-Mann stain.)

Digitized by Google

Digitized by Google

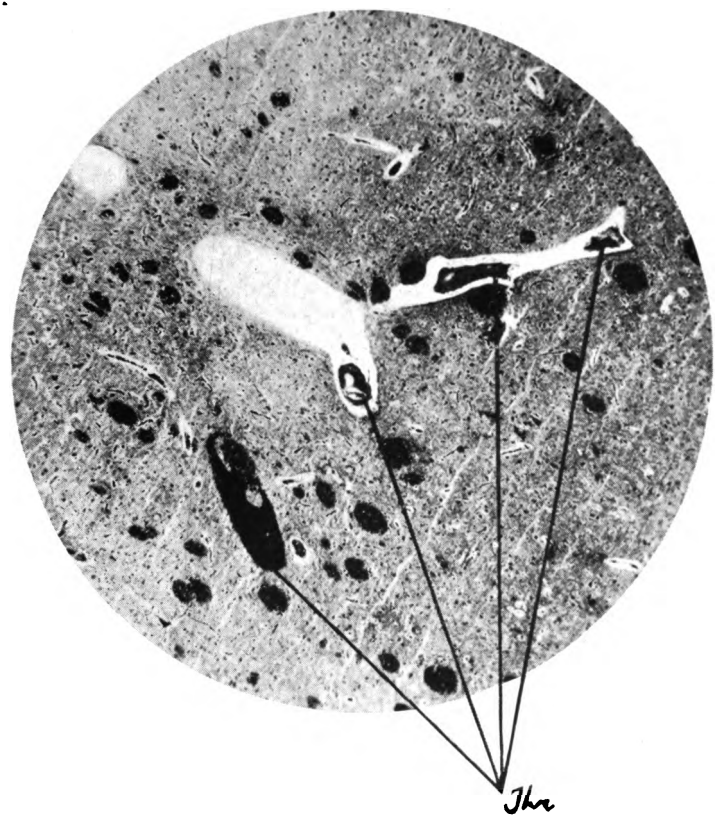

Fig. 13.—Nucleus candatus (its head bordering on the lateral ventricle). The numerous light channels inclosing vessels are so-called *Schrumpfräume* of Nissl; the large black spots are bundles of white cortical fibers *(Haubenfasern)* of the cortex; the small dots are glia nuclei. Some vessels show so-called hyaline thrombi (*Thr*). (Bielschowsky stain, × 50.)

Digitized by Google

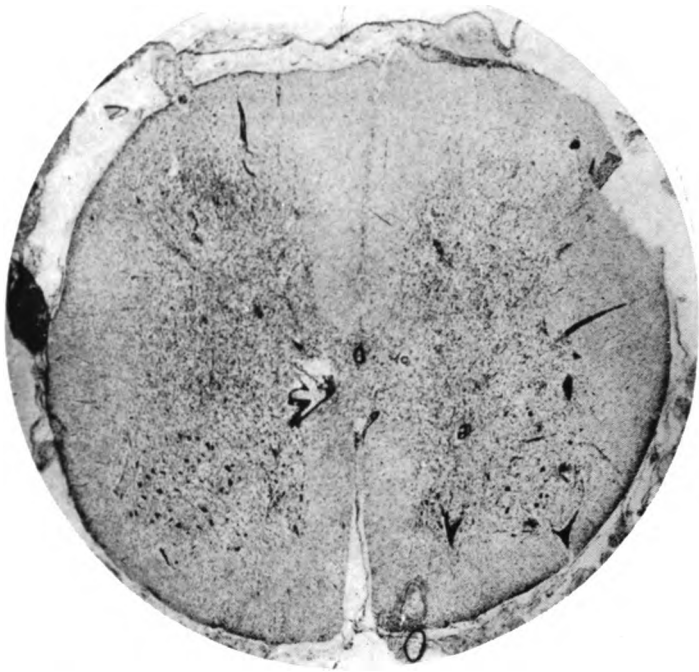

Fig. 15.—Lumbosacral portion of the spinal cord (Case 3). The membranes are slightly infiltrated; the subarachnoid space shows on the left a congested vein; the anterior and posterior horns exhibit a number of infiltrated vessels; the number of ganglion cells in the anterior horns is normal; the ependyma cells of the central canal are proliferated (a magnifying hand-glass will show the details better). (Toluidin-blue stain, × 16.)

Digitized by Google

CLINICAL SIGNIFICANCE OF THE CHANGES OBSERVED

The pial changes, as outlined, indicate a mild leptomeningitis, more marked in some portions than in others, but not so pronounced as in paralytic dementia or in poliomyelitis. However, the pial changes in their character and localization, though less intense, very much resemble those described and pictured by Mott,[4] Spielmeyer and others[5] in trypanosomiasis. Vascular changes are much less constant and much less pronounced in the cortex of the cerebrum and the cerebellum. Dozens of sections may not show a single markedly infiltrated vessel. The few cortical perivascular infiltrations seen are small and, like the pial ones, more numerous in the cerebellum. Nevertheless, the cerebral cortex shows an abundance of other pathologic changes. The architecture of the various cell layers is practically unaltered, though some regions such as the occipital lobe, do not appear as richly supplied with ganglion cells as normally.

CHANGES IN THE CORTICAL LAYERS

As a rule, the cortical layers contain a great number of glia cells, mostly in the form of so-called glia nuclei, as well as an abundance of capillaries. Some sections exhibit a rich capillary network, the vessels appearing much congested, but without any signs of sprouting, endothelial proliferation or perivascular infiltration. Their intima shows a well-stained endothelium, a free lumen not obscured or narrowed by a proliferated intima. The elastic membrane is homeogeneous, not split, powerfully developed and solidly blended with the intima. All these features are seen in Figure 4, which is taken from the pia whose ves-

4. Mott, F. W.: The Changes in the Central Nervous Systems of Two Cases of Negro Lethargy, Brit. M. J. **2**:1666, 1889. The Cerebrospinal Fluid in Relation to Diseases of the Nervous System, Brit. M. J. **2**: (Dec. 10) 1904. The Examination of the Tissues of a Case of Sleeping Sickness in a European, Brit. M. J. **1**: 1904. Histologic Observations on the Changes in the Nervous System in Trypanosome Infections, Especially Sleeping Sickness, etc., Arch. Neurol. **3**:581, 1907.

5. Franca and Athias: Les Plasmazellen dans les vaissaux de l'écorce cérébrale, dans la paralysie générale et la maladies du sommeil, Compt. rend. soc. de biol. **54**:192, 1902. Spielmeyer, W.: Die Trypanosomenkrankheiten, Jena, 1908; also, Schlafkrankheit und Progressive Paralyse, München. med. Wchnschr. **54**:1065, 1907.

Digitized by Google

sels are identical in appearance with those of the brain. Many capillaries, in Herxheimer (scarlet red) specimens, show minute and larger drops of fat enclosed within the adventitial cells, in some instances forming a solid ring around the lumen (Fig. 5).

Generally speaking, the cortical mesodermal elements show few pathologic changes. These are, however, quite pronounced in the ectodermal elements, namely, the glia and the ganglion cells. Some of the latter are of normal size and structure, but the majority appear rather homogeneous, uniformly stained, without Nissl bodies, and with a dark, pyknotic or very pale, granular nucleus. In some cells the disintegration goes so far that only the nucleolus is visible. The cell processes are mostly densely stained, homogeneous, tortuous and without any traces of Nissl substance. All types of cell changes described by Nissl are encountered, from the milder types, so-called chromatolysis and acute cell disease type, to the severe type when the cell body, including the nucleus, is reduced to an amorphous, granular mass invaded by glia cells—"neuronophages" (Fig. 6). Other less damaged cells are merely thickly surrounded by them (satellitosis). Satellitosis, chromatolysis and neuronophagia are the most common alterations to be met with in the cortex, especially in the deeper strata. In scarlet red specimens many cells, especially in the deeper layers, show droplets of fat mostly at the base of the cell, often surrounding the nucleus in the shape of a semicircle. The glia tissue, as we said, shows a marked proliferation of its cells, some of which have abundant protoplasm, but the majority are in the form of large nuclei rich in chromatin (Fig. 5). Astrocytes are absent, and glia fiber proliferation, as well as regressive changes, like ameboid glia, "fill bodies" (*Füllkörperchen* of Alzheimer), various granula and glia reticulum are totally lacking.

These parenchymatous changes are much more marked in the subcortical regions, the basal ganglia, subthalamic area and tegmentum. In these regions they are not only very pronounced, but they are also combined with profound interstitial changes. Thus, the vascular infiltrations are enormous (Figs. 7, 8, 9), in the case of some smaller arteries compressing their lumina, giving them the shape of a figure 8, or in the case of capillaries and small veins totally obscuring their shape (Fig. 9). The infiltration elements—lymphocytes, plasma cells, polyblasts, fibroblasts, rod cells—are principally confined to the adventitial

Digitized by Google

spaces of Virchow-Robin, but sometimes they are so dense that these spaces seem to be overflowed by the cells, and an impression is gained that the space outside the adventitia, between the latter and the brain—the so-called perivascular space of His—is also invaded. Careful observation, however, shows that the latter is usually free, and that the infiltration is exclusively confined to the Virchow-Robin spaces as well as to the distended meshes of the adventitia. In some places, like the optic thalamus and substantia nigra, the infiltration cells can be seen invading the parenchyma itself (Figs. 10, 11, 12), yet it is sometimes difficult to make out whether the invading elements (plasma cells and rod cells) traveled from a densely infiltrated vessel or are merely the focal products of inflamed or infiltrated minute capillaries.

CELLULAR ELEMENTS FREELY PRESENT

At any rate, some parts like the optic thalamus and substantia nigra, show a wealth of various cellular elements, including glia cells, all mixed in irregular masses, which surround vessels, capillaries and ganglion cells. Many vessels, especially the smaller ones, are not only densely infiltrated, but also congested, packed with blood pigment, some containing hyaline and other thrombi (Fig. 13). In spite of such marked infiltrative phenomena, some ganglion cells—especially in the substantia nigra — appear quite normal, though the majority show various changes (chromatolysis, neuronophagia and even cell sclerosis). As in the pia and cortex, hemorrhagic foci, gitter cells, foci of softening and polymorphonuclear cells are totally lacking. Only very minute capillary hemorrhages were found, and once a larger hemorrhage could be seen surrounding a congested small vein. In both instances, however, reactive phenomena in the neighborhood of the hemorrhagic foci were absent, and it is probable that these hemorrhages were terminal or agonal. In this connection, we should mention numerous red bodies—spherules—throughout the spinal cord of Case 3, in the gray as well as the white matter (Fig. 14). They are round and regular in shape, lying singly in the parenchyma or near the capillaries and even in the perivascular, adventitial spaces. They are smaller than red cells, are much more densely stained with eosin, are perfectly homogeneous and not enveloped by glia tissue. The latter is not thickened or proliferated around them. It is hard to tell at the first glance whether these bodies

Digitized by Google

are plain erythrocytes, or formations described as cystic plasma cells (cysto-plasmatozyta) by Perusini[6] and also depicted by Favorsky[7] in a case of tabes dorsalis. Still more do they resemble the bodies described by Fulci,[1] so it would appear proper to call them the *spherules of Fulci.* They show very well in Alzheimer-Mann specimens, appear in Bielschowsky specimens as round, pale brown bodies, and do not show at all with thionin, toluidin blue, hematoxylin-eosin or scarlet red stains, while in the brain tissue they cannot be demonstrated at all.

NO DEGENERATION PROCESSES OBSERVED IN SUBCORTICAL FIBERS

In spite of the marked ganglionic and interstitial changes, no degeneration of the subcortical fibers is found, nor are there any myelophages, gitter cells or other evidence of secondary glia changes. As in the cortex, the glia changes in the subcortical areas are of a progressive nature, represented by glia cells rich in protoplasm, or large-sized glia nuclei rich in chromatin. In the spinal cord there are, in addition, a great many astrocytes. The pons, medulla and spinal cord show the same intensity of interstitial changes as the large ganglia and midbrain. The spinal canal is usually occluded, either by proliferated ependyma cells, or by an amorphous mass mixed with lymphocytes and certain thin, elongated bodies stained dark with Bielschowsky's method (probably bacilli).

SUMMARY OF HISTOLOGIC STUDY

In summing up our histologic findings we may say that they concern the mesodermic (interstitial) and ectodermic (parenchymatous) elements. In the cortex, the parenchymatous changes predominate; in other regions (peduncles, internal capsule, lower portion of pons, bulb and cord) the interstitial changes predominate, while in the basal ganglia, substantia nigra and tegmental region of pons, both types are equally in evidence. In other words, the parenchymatous and interstitial changes may exist independently of each other, and an absolute relationship between them cannot be claimed.

---

6. Perusini, G.: Sopra Speciali Cellule Degli Infiltrati Nel Systema Nervoso Centrale, Riv. sper. di Freniat. **36**:721, 1910.

7. Favorsky, A.: Die Abbauerscheinungen bei Tabes Dorsalis, Nissl-Alzheimer's Arbeiten, **6**:74, 1913.

Digitized by Google

Both these large groups of pathologic changes are, according to Nissl and his school,[8] essential in and characteristic of an encephalitis. It is needless to state that the above-described findings would satisfy even such a severe critic as Nissl that our three cases are instances of an encephalitis, resembling as they do in their pathologic features paralytic dementia, sleeping sickness and poliomyelitis.

By the absence of foci of softening, of large hemorrhages, of gitter cells, of polymorphonuclear elements, the lethargic form of encephalitis, in our own cases at least, differs from other types, like Wernicke's polioencephalitis superior hemorrhagica,[2] acute hemorrhagic encephalitis,[9] influenzal encephalitis,[10] traumatic and experimental encephalitis (Friedman,[11] Coen,[12] Lottmar[13] and many others). In Wernicke's form and the so-called hemorrhagic encephalitis the hemorrhages are the principal feature with abundance of reactive phenomena on the part of the glia and total lack of parenchymatous, ganglionic changes. In fact, the characteristic signs of encephalitis, as outlined above, are absent in these two forms, which fact has been already pointed out by Biet,[14] Redlich[15] and Schroeder.[16] These authors justly doubt whether

8. Ranke, Otto: Beiträge zu der Lehre von Meningitis Tuberculosa, Nissl-Alzheimer's Arbeiten **2**: 1908.

9. Deiters: Ueber Haemorrhagische Encephalitis, Neurol. Centralbl. **17**: 722, 1898. Friedman: Deutsch. Ztschr. f. Nervenh. **16**:93, 1899. Brie: Neurol. Centralbl. **16**:2, 1897. Schmidt: Deutsch. med. Wchnschr. **18**:703, 1892. Sträussler: Jahrb. f. Psych. u. Neurol. **21**:253, 1902.

10. Bücklers: Arch. f. Psych. **24**:730, 1892. Königsdorf: Ein neuer Fall von acuter Haemorrhagischer Encephalitis waehrend der jetzigen Influenza-Epidemie, Deutsch. med. Wchnschr., 1892, p. 182. Fürbringer: Deutsch. med. Wchnschr. **18**:182, 1892. Oppenheim, H.: Deutsch. Ztschr. f. Nervenh. **6**:398. Oppenheim and Cassirer: Encephalitis in Nothnagel's Encyclop. **11**:2, 1907.

11. Friedman, M.: Studien zur Pathologischen Anatomie der acuten Encephalitis, Arch. f. Psych. **21**:461, 1890; also, Neurol. Centralbl. **8**:441, 1889.

12. Coen, E.: Ziegler's Beiträge zur Pathologischen Anatomie, etc. **2**:107, 1888.

13. Lottmar, Fritz: Beiträge zur Histologie der akuten Myelitis und Encephalitis, sowie verwandter Prozesse, Nissl-Alzheimer's Arb. **6**:248, 1913.

14. Biet: Contrib. a l'étude des affections nerveuses consecutives à la grippe, Paris, 1895. (Quoted from Friedmann, Footnote 9.)

15. Redlich, E.: Ueber Encephalitis Acuta, Centralbl. f. Allg. Path. **11**:513, 1900.

16. Schröder, Paul: Zur Lehre von der Akuten Haemorrhagischen Polioencephalitis Superior (Wernicke), Nissl's Arb. **2**:145, 1908.

Digitized by Google

Wernicke's polioencephalitis should be called encephalitis at all. The same can be claimed for the influenzal and other forms in which practically the only lesions are hemorrhages. In the traumatic and experimental forms, foci of softening or necrobiosis are common, which are, however, totally foreign to the lethargic type.

COMMENT

Our histologic studies thus have brought out the fact that the epidemic encephalitis distinctly differs from the condition previously described as influenzal encephalitis. Hence, if the influenzal encephalitis of previous authors really is influenzal, the form now under discussion decidedly is not. As stated before, it resembles the African sleeping sickness, dementia paralytica and the acute anterior poliomyelitis. The resemblance to the sleeping sickness is so great that a differential diagnosis on histologic basis is impossible. However, the sleeping sickness is chronic or subacute, while the lethargic encephalitis is rather acute in its course. The infiltrations of the pia and cortex are more pronounced in sleeping sickness. The slight difference in some pathologic changes (for instance, an occasional proliferation of vessels) may be explained by the longer duration of the African disease. It is also difficult to differentiate the brain changes of epidemic encephalitis from those of paralytic dementia. In the latter, however, the meningeal infiltration is very pronounced; the infiltration cells are mostly plasma cells, there is marked new formation of capillaries, a distinct proliferation of the intimal layer and of elastic fibers is present; all these changes being *especially pronounced in the cortex.*

As some of these characteristic features, like intima and elastica changes and proliferation of vessels, may be absent in acute cases of dementia paralytica and sometimes may be even more pronounced in the subcortical regions (the atypical forms of Lissauer and Alzheimer), it is clear that it may sometimes be difficult to differentiate these two diseases.

In poliomyelitis the changes are largely confined to the spinal cord, the cerebral forms being very rare. Suffice it to say, that the spinal cord changes of Case 3, though much resembling those of acute poliomyelitis (vascular infiltration), are by no means identical with them.

Digitized by Google

Hemorrhages, foci of softening, polymorphonuclear cells, marked satellitosis, so frequent and common in acute anterior poliomyelitis, are missing, while the parenchymatous and meningeal changes usually so marked in poliomyelitis are not striking in our case (Fig. 15).

The brief analysis of our findings shows a similarity in all essential points to those described in epidemic encephalitis by Marie,[17] Marinesco[18] and others.

CONCLUSIONS

1. The pathologic changes in epidemic (lethargic) encephalitis involve the entire central nervous system.

2. These changes are both parenchymatous and interstitial.

3. The former principally involve the cortex, the latter the peduncles, internal capsule, lower pons, bulb and cord. In the basal ganglia and midbrain both parenchymatous and interstitial changes are pronounced.

4. In its histopathology epidemic (lethargic) encephalitis differs from the various types of hemorrhagic encephalitis, but it is identical with that of African sleeping sickness (trypanosomiasis).

5. It differs from typical cases of paralytic dementia by the topography of the lesions and by absence of vascular wall changes, which probably are due to the more chronic course of the latter.

6. It also differs from the various types of traumatic and experimental encephalitis and from acute anterior poliomyelitis.

7. It is pathologically a distinct morbid entity, a disease *sui generis,* and does not resemble the conditions previously described as influenzal encephalitis.

8. The similarity between the pathologic changes in African sleeping sickness and epidemic encephalitis suggests a close relationship of their etiologic factors, that is, the epidemic encephalitis may be caused by a parasite akin to a trypanosome.

---

17. Marie, Pierre and Tretiakoff, C.: Examen histologique des centres nerveux dans deux cas d'encephalite lethargique, Bull. et mem. Soc. med. des hôp. de Par., May 24, 1918.

18. Marinesco, G.: Contribution a l'etude de l'histologie pathologique de l'encephalite léthargique, Bull. de l'Acad. de méd. **80**:411, 1918.

Digitized by Google

DISCUSSION

Dr. Max H. Weinburg, Pittsburgh: Speaking of the difference between what Dr. Bassoe found and the findings of the other workers such as Welsh and Marinesco regarding the differentiation of poliomyelitis and lethargic encephalitis, the findings are not in accord with those of the other workers. Marinesco pointed out that there are more plasma cells in the latter condition, and Welsh corroborated him. Marinesco pointed out that there is no neuronophagia or very little. This disease is of such wide manifestation that you will find a different pathology in the cases, just as clinically such cases manifest symptoms altogether different. There are many punctate hemorrhages found all over the brain. In one case we had in New York the lesion was believed to be a tumor. Most of the neurologists believed it to be a sarcoma of the brain. At necropsy a large hemorrhage can be explained readily by the fact that the disease in some cases is more marked than in others, and therefore there is some variance in results. Welsh reported several cases of the cord where there were cervical lesions, but in one case the lesion was as far down as the lumbar region. Clelland of Australia who has experimented on 120 animals points out mainly the perivascular infiltration and that the disease can be transmitted. The differential points between poliomyelitis and encephalitis lethargica are that there is no or little neuronophagia as there is in poliomyelitis, the plasma cells predominate, and there is little parenchymatous change, and it is for that reason that most of these patients recover. There is a prevailing belief that this is a disease of high mortality, but this is merely due to the fact that a great many cases are not recognized. I have had occasion to see over sixty cases in New York with a very low mortality.

Dr. Peter Bassoe, Chicago: In regard to the question of cell changes, I can only relate my own experience. At the time I reported my first two cases I stated that hemorrhage was marked and there were no cell changes. After making a thousand checks we found the hemorrhage was an accidental finding. I happened to strike the only hemorrhage the first time. You must make sections all through and conduct your investigation with an open mind. I find hemorrhages are not a feature and neuronophagia is. It is, to be sure, not overly profound. You can look at a dozen sections, particularly of the cortex, without finding it, but deeper down you will not miss it. I have now a brain from a patient who had an acute encephalitis, and there is an extensive condition throughout the brain. It may be a true influenzal encephalitis. I am not prepared to say anything about the relation of this disease and influenza; only if what has previously been described is influenzal encephalitis, then this is not. As to the Australian cases, we have to be a little slow, as we do not know what they were. Many of the reports look like poliomyelitis, and one of the foremost investigators there believes the cases were poliomyelitis.

Digitized by Google

THE RÔLE OF THE PITUITARY GLAND IN EPILEPSY

BEVERLEY R. TUCKER, M.D.
Professor of Neurology and Psychiatry, Medical College of Virginia
RICHMOND, VA.

The problem of epilepsy may be best approached by considering all convulsions which are accompanied by unconsciousness, whether periodic or otherwise, as symptoms of some underlying pathologic state or disease. By doing this the convulsion becomes a symptom. The question then arises, which I believe should be answered in the negative, whether any so-called epileptic state should, of itself, be considered a disease.

ETIOLOGIC FACTORS

Thus, among the underlying causes of convulsions are uremia, eclampsia and other toxicoses; several varieties of trauma, as birth trauma and that from blows on the head; certain intracranial irritations, as brain tumors and cerebral hemorrhages; certain infections, as syphilis and varieties of meningitis; certain conditions of cerebral maldevelopment accompanied by a degree of brain cell deterioration; *and certain conditions of the pituitary gland causing a change in its secretion.*

I believe, therefore, that convulsions, whether periodical and termed epilepsy, or not, are due to definite pathologic cerebral tissue changes which may be congenital or induced, and hence, that epilepsy is an organic and not a functional condition. In the toxic and pituitary cases there may exist previous malformation and instability of the cortical cells, or, on the other hand, it is perfectly conceivable that the toxic substance or the changed pituitary secretion may produce their own cortical cell deterioration. I published a paper on the relation of the pituitary gland to some cases of epilepsy, and in 1914[1] and again in 1916,[2] a paper on the subject of hypopituitarism in its relation to

1. Tucker, B. R.: South. M. J. **7**:608 (Aug.) 1914.
2. Tucker, B. R.: Virginia M. Semi-Month. **31**:1 (April 7) 1916.

Digitized by Google

epilepsy. These papers were the result of a personal suggestion of Dr. Harvey Cushing and also of the perusal of his most excellent book.[3]

Dr. Daniel D. Talley has roentgenographed most of my cases and in nearly all instances the roentgenographic findings have confirmed the clinical diagnosis of either the permanent or the transitional stage of hypopituitarism. Dr. Johnston of Pittsburgh, I understand, was one of the earliest roentgenographers in noting pituitary changes in certain cases of epilepsy. In a paper read[4] before this section in 1918 I described transitory pituitary changes, causing psychoses occurring during adolescence. Some of these transitory changes appeared also to be the cause of convulsive states. Cushing believes that the secretion of the posterior lobe of the pituitary joins the cerebrospinal fluid and thus bathes the cerebral cortex with a substance which is necessary to the functional stability of the cortical cells. When this secretion is diminished or absent the cells may be said to deteriorate. Whether this deterioration is due to the lack of whatever the pituitary secretion may give them, or whether it takes place by the lack of this substance allowing toxic substances to exert a deleterious influence, I am not sure. At any rate it appears that the administration of pituitary gland extract tends to alleviate symptoms caused by undersecretion of the pituitary gland.

TWO TYPES OF CASES

There are two forms of hypopituitarism here described: one chronic or congenital, and the other transitional. In *chronic or congenital types,* the patients have, among other indications, increased fat, lack of body hair, feminine distribution of pubic hair in the male, scanty menstruation in the female, diminished perspiration, increased sugar tolerance, small genitalia, and frequently bradycardia and lowered blood pressure. In the *transitional type* the patient may show evidence of normal pituitary secretion, or hypersecretion in the past, which during adolescence becomes lessened. This is at times traceable to some illness or trauma. In these cases the patient begins to take on

3. Cushing, Harvey: The Pituitary Body and Its Disorders, Philadelphia, W. B. Saunders Company.

4. Tucker, B. R.: Pituitary Disturbance in Its Relation to the Psychoses of Adolescence, J. A. M. A. **71**:330 (Aug. 3) 1918.

Digitized by Google

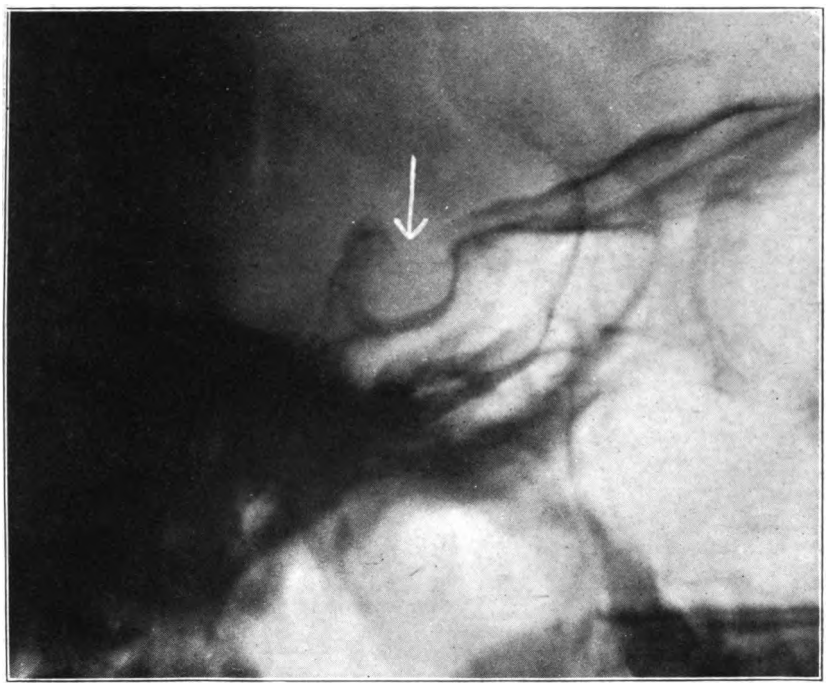

Fig. 1.—Large fossa, elongated processes slightly roughened; hyperpituitarism.

Digitized by Google

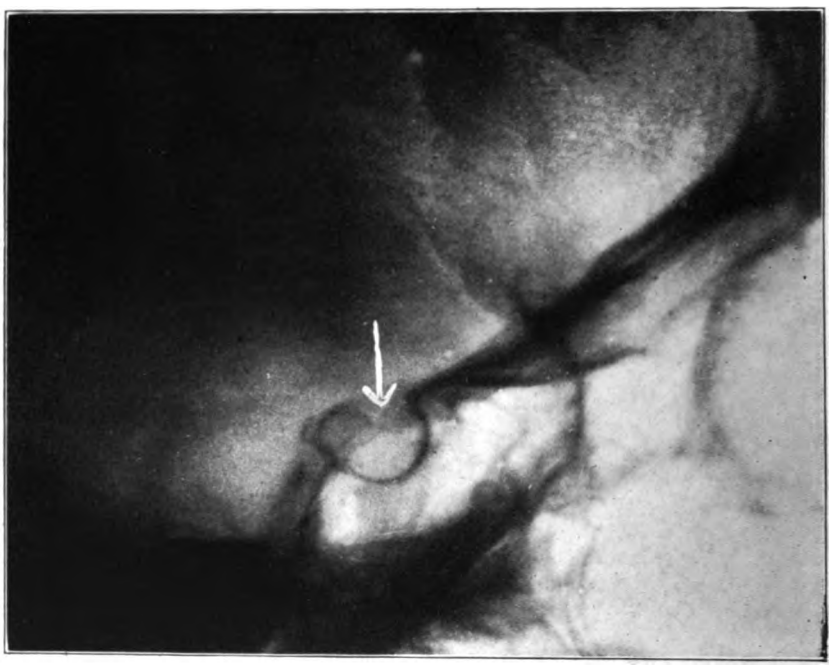

Fig. 2. — Small fossa, anterior and posterior processes approximated hypopituitarism.

Digitized by Google

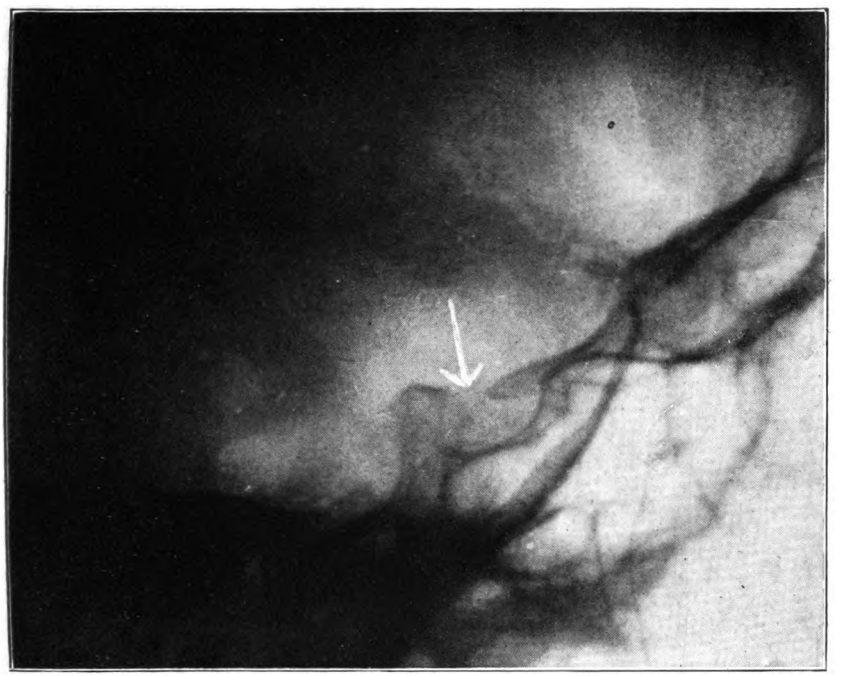

Fig. 3.—Moderate-sized fossa, enlarged roughened processes; transitional case.

Digitized by Google

Digitized by Google

flesh, has an increased desire for sweets, become less energetic, eats voraciously, perspires less and may have slow pulse rate and lowered blood pressure. In either type occasional or periodic convulsions may occur, which usually make their appearance during adolescence. Convulsions may also occur in cases of tumor of the pituitary body, as pointed out by Cushing, but the discussion of tumor cases is not included in this paper.

TABLE 1.—CHRONIC HYPOPITUITARY TYPE *

Case	Age of Onset of Attack	Type of Attack	Average Frequency of Grand Mal	History of Injury or Disease	Preparation of Gland Used	Result of Feeding	Remarks
1	11	Grand	1 a month	Injury to head when 7 years old	Whole gland	Two in 18 months	Marked improvement
2	14	Grand	1 every month	None	Whole gland	None in 3 years	Cured
3	18	Grand	1 every 6 weeks	Head injury as child	Whole gland	None for 14 months	Attacks ceased
4	18	Grand	1 every 2 months	None	Anterior lobe gland	1 every 2 months	Not improved
5	11	Grand	1 a month	None	Whole gland	Three in 18 months	Marked improvement
6	12	Grand	2 to 3 a week	None	Whole gland	No attacks over 3 years	Cured
7	10	Petit	No grand, petit daily	Injury to head as child	Whole gland	No attacks over 3 years	Cured
8	13	Petit	No grand, petit many a day	None	Whole gland	None for over 3 years	Cured
9	12	Grand	3 a year	None	Whole gland	None for over a year	Prospects of a cure
10	13	Grand and petit	1 to 2 a week	Head injury as a child	Whole gland	One a month	Improvement
11	9	Grand	3 or 4 a year	Head injury 5 years old	Anterior gland	None for 2 years	Prospects of cure
12	30	Grand	1 every 6 weeks	None	Whole gland	One in 2 years	Prospects of cure
13	17	Grand	3 a year	Malaria	Whole gland	Same as before	Took medicine irregularly
14	15	Grand	1 a week	None	Whole gland	One in 2 months	Marked improvement
15	13	Grand	1 every 6 weeks	Rheumatism	Whole gland	One every 3	Improvement
16	11	Grand	1 a month	None	Whole gland	None for over 2 years	Apparent cure
17	8	Grand	1 every month or 2	None	Whole gland	None for 6 months	Marked improvement

* All of these patients had definite evidence of clinical hypopituitarism and roentgenographic signs of the chronic type of hypopituitarism. The general health and mental condition of the patients improved while taking pituitary extract.

PATHOLOGIC CHANGES OBSERVED

Roentgenographic examinations in the chronic or congenital type of hypopituitarism have shown small fossae with large clubbed posterior processes and frequently elongated anterior processes tending to

Digitized by Google

bridge the fossa. Many of these cases have shown irregularity in the contour of the posterior part of the sella.

In the transitory type roentgenographic examinations have revealed either normal-sized or enlarged fossae, according to whether the clinical symptoms have pointed to normal or hypersecretion in the past. The posterior processes have been enlarged and clubbed. The anterior process may be enlarged and, at times, bridging between the processes has been observed. Roughening of the posterior contour of the sella is usually found.

TABLE 2.—TRANSITIONAL HYPOPITUITARY TYPE *

Case	Age of Onset of Attack	Type of Attack	Average Frequency of Grand Mal	History of Injury or Disease	Preparation of Gland Used	Result of Feeding	Remarks
1	18	Grand	1 every 2 months	None	Anterior lobe gland	One attack 18 months	Marked improvement
2	25	Grand and petit	1 every 2 months	None	Whole gland	Recent	Too recent to judge
3	13	Grand	1 every 3 weeks	Injury to head as child	Whole gland	Two in 9 months	Marked improvement
4	24	Grand	1 or 2 a month	None	Whole gland	Same as before	No improvement
5	20	Grand	1 every 6 months	None	Anterior lobe gland	None for 2 years	Prospects of cure
6	12	Grand and petit	1 a week	None	Whole gland	None for 18 months	Prospects of cure
7	17	Grand	1 every 2 or 3 months	Injury to head as child	Whole gland	One series of several attacks	Improvement
8	17	Grand	2 to 4 a week	None	Whole gland	No attacks for 8 mo.	Prospects of cure
9	22	Grand	1 a month	None	Whole gland	One every 2 months	Improvement
10	22	Grand and petit	3 a week	Injury to head few years before	Whole gland	None for 18 months	Prospects of cure, petit attacks rare
11	13	Grand	1 a week	None	Anterior lobe gland	One every 2 months	Marked improvement

* All of these patients had definite evidence of clinical hypopituitarism and roentgenographic pictures revealed the transitional type of that condition. It was noted that the general health and the mental condition of the patients greatly improved in all cases.

The roentgenographic difference, therefore, between the two types lies chiefly in the size of the fossae, that of the chronic type being decreased and that of the transitional type being normal or enlarged in general contour even if encroached on by bony growth.

AUTHOR'S OBSERVATIONS

The present investigation is based only on the study of my last 200 cases of epilepsy, for the reason that I do not feel that previous to these cases I had knowledge or experience enough in the pituitary

Digitized by Google

type of convulsions to examine them sufficiently or judge of them accurately.

Of the 200 cases, sixty-three, or 31.5 per cent., revealed some evidence of pituitary disturbance. Of these sixty-three cases, thirty-five were discarded for the reason that they had in addition to clinical and roentgenographic signs of dispituitarism, evidence of syphilis, marked nephritis, active toxemia from focal infections, hydrocephalus, organic brain disease or injury, or because the evidence of pituitary disturbance was so slight that it was negligible. However, in most of these, pituitary feeding was beneficial. We thus have left *twenty-eight pure pituitary cases, or 14 per cent.* Of these twenty-eight cases, seventeen were of the chronic hypopituitary type, and eleven of the transitional hypopituitary type.

TREATMENT

In the treatment of these cases if bromid was being taken small doses were continued, and in some of the severe cases small doses were given, especially at first. The effort, however, was made to get the patient off of bromid.

CONCLUSION

I believe that there is a definite relation between the undersecretion of the pituitary gland and a group of periodic convulsive attacks usually termed epilepsy; that this group is divided into a chronic hypopituitary type and a transitional hypopituitary type by both clinical and roentgenographic evidence; and that pituitary gland feeding has a markedly beneficial effect not infrequently leading to cure.

DISCUSSION

Dr. Walter Timme, New York: Dr. Tucker's report on what he has done is perhaps really too modest. The number of phases we see of this type that can be helped as far as the epilepsy is concerned, is quite large. The cures are cures as effective and quite as prompt as he has suggested. It remains to determine which are the proper cases for such therapy. You cannot from the appearance of the sella turcica on the roentgenogram determine if the person has epilepsy. Every hypopituitary case in which the patient has epilepsy shows sella changes, but every case with sella changes does not necessarily mean that the individual has epilepsy. You must remember also that the formation of the sella turcica does not necessarily go hand in hand with equivalent changes in the hypophysis. If the sella is large you can have all kinds of changes in the

Digitized by Google

hypophysis without any effect on the sella. Sir Victor Horsley made an interesting experiment in this direction. He found the cerebral cortex in dogs was more sensitive to electric currents after removal of the hypophysis than before. That led him to suspect that the irritability of the cortical cells was in a measure due to the lack of secretions of the hypophysis. As a matter of fact, these hypopituitary individuals, even though apparently sluggish, have also increased cortical irritability. They improve by feeding on pituitary extract. In Plattsburg we saw a man who had epileptic convulsions and the case was recognized as one of dyspituitarism of the Froelich dystrophia type. He was put on pituitary treatment and thereafter had only one convulsion in six months, previously having had convulsions daily, or three or four times a week. I have two slides which, with the permission of the chairman, I will show, demonstrating this phase of the situation. They are both cases of dyspituitarism, but show absolutely different physical signs. The acromegalic had epilepsy, the Froehlich dystrophia type had not. The acromagelic was cured by administration of pituitary substance.

Dr. H. H. Hoppe, Cincinnati: I have been struck with the relation between certain types of dyspituitarism and the appearance of epilepsy, especially the type which shows changes of a sexual character in young individuals. I had one case in particular, a boy who had feminine characteristics and large masses of fatty distribution. He has been under treatment four or five years with extract of pituitary gland, and there has been no change in the number of attacks or in the character of the epileptic attacks as a result of the administration of pituitary gland. Perhaps I made the mistake of limiting him to the anterior lobe. The anterior lobe stands in close relation with the development of sexual characteristics and if you have disease of the anterior lobe you will have heterogeneous sexual characteristics. This boy showed typical feminine characteristics, and the change that has occurred in him has been the development of characteristics of a masculine nature. He has grown to be a strong, slender, handsome man, all the feminine characteristics have disappeared except the masses of fat, but the epileptic attacks have not been changed by the administration of the anterior portion of the gland. I consider it was the dyspituitarism which gave rise to this case and to the abnormal sexual development. I have another case in which there has been no result from treatment, perhaps on account of the same limitation to the anterior lobe.

Dr. E. B. Angell, Rochester, N. Y.: I wish to corroborate the statement made by Dr. Tucker and assure Dr. Hoppe that if he would use the extract of the whole gland he would get results. I believe it is the cases that occur during puberty that receive the most benefit from this therapy. I have been following the same process in a mild form of dementia praecox and have obtained similar results. I have had several remarkable results with extract of the whole gland in several cases of dementia praecox with the very type that presents some of these symptoms at puberty.

Dr. George A. Moleen, Denver: I believe we are wise to believe that epilepsy and dementia praecox are produced by the same conditions. I think

Digitized by Google

we all realize that when we find a disproportional secretion of the endocrine glands we are entitled to consider this as due to disequilibration in the same fashion as the hormones are considered. I do not see why we cannot associate a form of epilepsy to the hormonic inequilibration any more than we can associate epileptic seizures with alterations in the sella turcica. We find many patients with pituitary evidences predominating, but more are without epilepsy than with. That is particularly true in the normal type of preadolescent deficiency. We are able to interpret them as the result of hormonic disequilibration rather than as disease of one of the structures, namely, the pituitary itself.

Dr. Albert E. Sterne, Indianapolis: Some years ago I used the anterior lobe of the pituitary in a large number of cases of epilepsy and had no results whatever. I came to the conclusion that it was absolutely ineffective and have seen no reason to change that opinion since. I have had no beneficial results whatever in the administration of the anterior lobe of the pituitary. Dr. Timme said yesterday that the anterior lobe extract was rather dangerous. My experience has been quite the contrary. He stated that in using the whole pituitary he used fresh substance. I would like to know where he obtains the fresh gland. We have had difficulty in getting the substance of any of the gland. The general results with feeding of the whole substance formerly was appreciably better than with the anterior or posterior lobes alone, but I cannot say that our results in several hundred epileptics have been anything like as pronounced as the report of the essayist, Dr. Timme and others. Either the substance they used is different or else they have had better success in the administration. There is another point I would like to mention, in reference to the roentgenograms of the sella turcica that were shown by Dr. Timme. They were all taken from the lateral point of view. Recently we have been taking our roentgenograms from the top directly so the rays enter through the infundibular opening. That gives us the dimensions from side to side. We have found that the sella turcica thus shows up better. We sometimes think we are dealing with an imprisoned hypophysis when we are merely judging from a side view.

Dr. Walter Timme, New York: With the permission of the chairman I will answer the question regarding the extract of the pituitary substance. I usually have a man go to the abattoir and get the whole substance. He dries it and puts it in capsules. Occasionally we use the steer, but usually the hog. The Armour preparation is fairly fresh, and if taken within a reasonable length of time and kept cold and dry it will answer. Regarding the roentgenograms I showed, they were taken almost directly laterally and always stereoscopically and one directly down, but only one picture was shown on the screen which gave the general structural outline to the best advantage.

Dr. Irving J. Sands, Brooklyn: In regard to the roentgenographic changes in the sella turcica I have tried to check them up at necropsy in fifty cases and I have not found any relation between the pituitary gland and the size or shape of the sella. We have had roentgenograms made of a few of these

Digitized by Google

patients antemortem, and there was no relation between the changes in the pituitary and the sella turcica. I believe that with a few exceptions, such as acromegaly, the sella has no more to do with the condition or function of the pituitary gland than the shape of the nose has to do with one's respiration. I believe that the fibrous condition of the capsule has more to do with the functional possibilities of the pituitary gland because we have found many changes in the capsule where there were none in the sella itself.

Dr. Beverley R. Tucker, Richmond, Va.: I think the shape of the nose has a good deal to do with the respiration. As far as the glandular substance is concerned, there are few patients to whom I give the anterior lobe, particularly if the blood pressure happens to be high. I give the whole gland capsule. There are three elements that must be present in these cases. If you want to have results you have to have the age of adolescence, the clinical type and the roentgenographic findings. It is very hard to get a necropsy on an epileptic, but I would not take the finding on a case that did not have convulsion as a criterion at all. There are two distinct types of chronic hypopituitary cases. It is usually after puberty that these convulsions begin. Some give a history of having been normal until receiving a blow on the head or suffering from an acute infection after which the convulsions began. It seems to stir up something in the gland.

I remember ten or twelve years ago a neurologist made the statement before our Section that there was no such disease as neurasthenia. We were shocked then, but I think we have all come around to his opinion today. We may as well assume now there is no such disease as epilepsy which is always due to some distinct pathologic condition, and we will thereby separate off one group after another and treat the convulsions as a symptom, and cure the condition incidentally. Looking over these 200 cases, I found that ten were syphilitic; they had positive Wassermann reactions. These people should not be considered epileptics; they should be considered syphilitics. If we take that view we will approach the passing of the old hopeless epilepsy.

Digitized by Google

THE RESEMBLANCE OF THE SENSORY SYMPTOMS OF POST-DIPHTHERITIC ATAXIA TO THOSE SEEN IN THE CORD CHANGES OF SEVERE ANEMIA*

GEORGE WILSON, M.D.
Instructor of Neurology, University of Pennsylvania; Assistant Physician to the Philadelphia General Hospital
PHILADELPHIA

Combined sclerosis or postero-lateral sclerosis of the spinal cord occurring in certain conditions, more especially pernicious or grave secondary anemia, has been recognized for many years; it was not, however, until 1913 that the characteristic loss of sensation was pointed out by Dejerine.[1] That eminent French neurologist showed that the affection of the posterior columns begins medially and spreads outward; as a result the fibers which are first involved are the long conduction fibers which ascend uncrossed in the columns of Goll and Burdach. This gives a loss of certain forms of deep sensibility, that is, of the sense of position and the sense of vibration; all other forms of sensation remain intact until the disease has produced widespread changes in the cord, changes which amount practically to a transverse myelitis. Patients with the disease almost invariably have subjective sensory disturbances in the form of paresthesias of the feet, legs and hands.

Diphtheria may involve the nervous system in a number of ways, thus the only sign that nervous structures have been attacked may be paralysis of the soft palate or of accommodation. The disease often produces serious disturbances of motion and coordination in the extremities. There is often a diffuse weakness or paralysis of the lower or upper extremities or of all four extremities. Another manner in which diphtheria may involve the nervous system is in the produc-

* From the Department of Neurology, University of Pennsylvania.

1. Dejerine: Société de Biologie **75**:554, 1913.

Digitized by Google

tion of an acute ataxia which Dejerine[2] has described as a peripheral neurotabes.

From a pathologic point of view there is widespread involvement of the nervous system in diphtheria. Oppenheim states that "the diphtheritic poison acts upon the whole nervous system, producing the most marked symptoms now at this site, now at that, but most often in the peripheral nerves, and that it may also have a toxic effect upon certain areas without producing structural changes in them." He also states that the neuritic processes constitute the essential basis of diphtheritic paralysis. That this is true of post-diphtheritic ataxia I seriously doubt.

LITERATURE

A number of investigators have examined the nervous system in diphtheria. Some have studied material from persons who died after an acute illness, some from patients who died weeks or months after the attack of diphtheria and others have approached the subject from an experimental angle.

Preisz[3] found a marked degeneration of the peripheral nerves. In the cord he found numerous hemorrhages, chiefly in the gray matter. The white substance was normal, except in one case which presented an advanced grade of degeneration in the posterior columns. Bikeles[4] in one case of post-diphtheritic paralysis and ataxia found degeneration of the posterior column and to a lesser degree of the posterior roots. Arnheim's[5] cases showed extensive degeneration of all nerves examined and capillary hemorrhages in the cord. Thomas[6] found degeneration of the peripheral nerves, and in the cases where the cord was examined and in which a definite pathology was present, the posterior columns were always most seriously affected.

Henschen[7] reported a case of acute disseminated sclerosis following diphtheria. His patient died three months after the attack. The microscopic study of the nervous system showed, in addition to a

2. Dejerine: Arch. d. Physiol., 1887.
3. Preisz: Deutsch. Ztschr. f. Nervenh. **6**:95, 1895.
4. Bikeles: Wien. **2**:26, 1894.
5. Arnheim: Arch. f. Kinderh. **13**:461, 1891.
6. Thomas: Reports of the Boston City Hospital, 1898.
7. Henschen: Fortschritte der Medicin, p. 529, 1896.

Digitized by Google

marked polyneuritis, a great number of disseminated areas of sclerosis in the cord, most marked in the posterior columns and associated with intense perivascular infiltration. Oppenheim[8] and Redlich[9] have had similar cases. In experimental work, Babes[10] found general edema and softening in the cord most marked in the posterior and lateral columns. Martin[11] found the cord normal in his experiments.

There have been a number of clinical reports which show that various parts of the central nervous system may be involved during an attack of diphtheria. Rolleston[12] investigated the plantar reflex in 870 cases of diphtheria. In 172 of these the extensor response was obtained at some period of the illness showing that the pyramidal tracts were involved either through a toxic action or by edema and capillary hemorrhage. The mortality was higher in the class of patients who developed the Babinski reflex and the incidence of paralysis was greater.

Taylor[13] reported a case of Friedreich's ataxia which developed after a post-diphtheritic paralysis. In analyzing the symptomatology of that case it seems to me more likely that his patient really never recovered from the diphtheritic involvement of the central nervous system.

Tinel and Siredey[14] reported the eighth case in literature of chronic bulbar paralysis following diphtheria. They explained the occurrence of the bulbar symptoms in their case by assuming that the toxins traveled along the nerves and ultimately produced sclerosis of certain nuclei of the cranial nerves.

AUTHOR'S OBSERVATIONS

The cases which I desire to record are of interest from the type of sensory loss which they show, the loss of sensation being exactly

8. Oppenheim: Textbook of Nervous Diseases, Chicago, Chicago Medical Book Co. **1**: 321.

9. Redlich: Die Deutsche Klinic, p. 561.

10. Babes: Virchows Arch. f. path. Anat. **119**:168, 1890.

11. Martin: **1**:26, 1892.

12. Rolleston: Rev. Neurol. and Psych., p. 404, 1910.

13. Taylor: Proc. Roy. M. & Chir. Soc., Sec. of Neurol. **7**: 1913-1914.

14. Tinel and Siredey: Bull. et mém. Soc. méd. d. hôp. de Par., Nov. 22, 1906, p. 1126.

Digitized by Google

similar to that seen in the combined sclerosis of pernicious or severe secondary anemia. The subjective symptoms which were complained of in these cases were of the same character that is so common in anemia.

REPORT OF CASES

CASE 1.—*A man, aged 22, had diphtheria two months before the onset of symptoms which were paresthesia in hands and feet, fatigue on exertion and difficulty in walking. Examination revealed a Romberg sign; loss of all deep reflexes; loss of the sense of position and vibration in feet and legs; marked impairment in recognising the points of a compass; all other forms of sensation were intact.*

History.—The patient, aged 22, a student in the University of Pennsylvania, came to the Students' Dispensary in the last week of February, 1917. During Christmas week of 1916, he had diphtheria, and was given three injections of antitoxin during the course of the illness which confined him to his room for ten days. He was apparently well until a few days before he came to the dispensary, two months after the attack of diphtheria. At that time he had numbness in the feet and hands. The numbness became gradually worse and was associated with other paresthetic phenomena. The legs became weak and walking caused fatigue. He did not have either difficulty in reading or diplopia.

Examination.—The gait was ataxic and a Romberg sign was present. The biceps, triceps, patellar and Achilles reflexes were absent. Plantar stimulation produced flexion of the toes on both sides. The extensors of the toes and feet were slightly weak. Sensation was normal for light touch, pain (pin prick and the pain of deep pressure), heat, cold and deep pressure throughout the body. The sense of position was seriously disturbed in the toes and ankles; the vibratory sense was affected below the middle of the legs; there was impairment of the compass test over the feet and lower part of legs. There was slight ataxia and clumsiness in the upper extremity, without any disturbance of sensation or motion. The cranial nerves were normal. The nerve trunks were not tender on palpation. This man recovered entirely and when last heard of was with the American Expeditionary Force.

CASE 2.—*A soldier, aged 24, contracted diphtheria in France early in January, 1918; shortly after his throat cleared up, he had difficulty in reading; regurgitation of fluid from the nose; six weeks later paresthesias in the feet, soon involving the legs and hands; marked ataxia in all extremities; Romberg sign present; all deep reflexes lost; sense of position, vibratory sense and the compass test markedly disturbed in the feet, legs and hands; all other forms of sensation intact.*

History.—This patient was a soldier belonging to the 163d Infantry and stationed near Blois, France. In the first week of January, 1918, he had a severe attack of diphtheria for which he was treated at the camp infirmary for two weeks. Coincident with his discharge from the hospital he had dim-

Digitized by Google

ness of vision and regurgitation of liquids through the nose. These conditions persisted three weeks and then gradually wore away. On February 20, he had for the first time paresthesias in the feet; the paresthesia became worse and soon involved the legs and hands. In a short time he had difficulty in walking and in using the hands for the finer movements, such as dressing and writing. He had no pain. He was sent to Blois on March 28.

Examination.—He was very ataxic in the finger to nose test and in walking and presented a marked Romberg sign. The biceps, triceps, patellar and Achilles reflexes were lost. Plantar stimulation produced flexion of the toes. There was no motor involvement with the exception of slight weakness in the flexors of the fingers and wrists. The cranial nerves and the pupillary reflexes were normal. Sensation was normal for light touch, pain (pin prick and deep pressure), heat, cold and deep pressure throughout the body. The sense of position was seriously impaired in the toes, ankles, fingers and wrists; the vibratory sense and the compass test were impaired over the feet, legs and in the fingers and hands. The nerve trunks were not tender on pressure. This patient was not followed up.

CASE 3.—*A woman, aged 37, had diphtheria in November, 1917; at Christmas, paresthesias in feet which in time involved legs and hands; difficulty in walking and in performing fine movements with hands; gait and station markedly ataxic; sense of position and vibration impaired in toes and feet; touch sense very slightly impaired over dorsum of feet; all other forms of sensation normal.*

History.—This patient was referred to Dr. Spiller by Dr. Guthrie of Sayre, Pa., and she was admitted to the University of Pennsylvania Hospital, Feb. 18, 1918. I am indebted to Dr. Spiller for the privilege of reporting this case and the following notes are taken from the hospital record. In November, 1917, she had diphtheria and was ill for two weeks. She was apparently well from the first day out of bed until Christmas, when paresthesias were noted in the feet. In the course of three weeks the paresthetic sensations ascended as high as the knees. Two weeks before admission to the hospital, she began to have difficulty in walking and in using the hands for sewing and writing. She had never had pain in the legs or arms.

Examination.—The gait was very ataxic and a marked Romberg sign was present. There was no ataxia in the finger to nose test. The biceps, triceps, patellar and Achilles reflexes were absent. Plantar stimulation produced flexion of the toes on both sides. The arms and hands were moved about freely and no muscular weakness was present The lower extremities were moved well but there was slight weakness in certain movements, namely, extension of the toes and feet and extending the legs on the thighs. Sensation was normal for pain, heat and cold throughout the body. Touch was slightly impaired over the outer side of the dorsum of both feet. The sense of position was lost in the toes of both feet, but was normal at the ankles. The sense of vibration was lost over the feet, and was diminished over the lower part of the legs. The nerve trunks were not tender on deep pressure. The cranial nerves, the reflexes of the pupils, the blood and the cerebrospinal fluid were normal. The

Digitized by Google

urine showed a slight amount of sugar and on one occasion a positive test for acetone. This patient remained in the hospital only four days.

PRACTICAL CONSIDERATIONS

From an analysis of the sensory symptoms in these cases it will be noticed that all three cases had paresthesias in the hands and feet, and all showed a loss or marked impairment of the sense of position and of vibration, and that in two of the cases with absolute preservation of touch sense there was impairment of the ability to recognize the two points of a compass.

Cassirer[15] has reported a case of post-diphtheritic polyneuritis with loss of osseous sense and the sense of position.

Where is the pathology of a process which can pick out and impair the conduction fibers for the sense of position and the osseous sense, and also render the recognition of the two points of a compass impossible or nearly so?

Head[16] has given us the following information regarding the relation of the various forms of deep sensibility (with the exception of the sense of vibration) to one another in the peripheral nerves and in the spinal cord and also their relation to the compass test. The power of discriminating two points of a compass depends on the integrity of touch sense. If this is impaired it is clear that the compass test is of little use. In cord lesions studied by Head, especially cases of Brown-Séquard paralysis, the power of discriminating the two points of a compass was impaired or lost on the side of the motor weakness and was associated with loss or impairment of the sense of position and passive movement. In the peripheral nervous system there is an intimate relation between sensibility to deep pressure and the pain produced by it, and the ability to recognize passive movement and position. All three are present or absent after lesions of the peripheral nerves and all three depend on the integrity of those fibers which run with the muscular nerves. In the spinal cord the fibers conducting the sense of position and passive movement are separated from those conducting deep pressure and the pain of deep pressure and ascend with the vibratory or osseous sense in the posterior columns.

15 Cassirer: Die Deutsche Klinic, p. 804.

16. Head: Brain **26**:537, 1906.

Digitized by Google

LOCATION OF THE LESIONS

It seems reasonable to me to place the lesions which account for the sensory loss in the cases here described in the posterior columns of the cord and not in the peripheral nerves. So far as I know, in no other part of the nervous system are the fibers which conduct the osseous sense, the sense of position and the ability to recognize the points of a compass grouped together. In all of these cases the motor loss was insignificant and the ataxia was the predominating symptom. If disease of the peripheral nerves were responsible for such a loss of sensation one would expect to find in addition, a loss of deep pressure sense and a marked impairment of motor power.

Pathologic reports show that the posterior columns of the cord are usually most seriously damaged when the cord is involved during the attack of diphtheria. The cases of Bikeles, Henschen, Oppenheim and others show the same to be true in cases that recover from the acute illness only to succumb to the subsequent disease of the nervous system. In the consideration of the changes which occur in the central nervous system during an attack of diphtheria it is interesting to note that perhaps a number of cases of disseminated sclerosis may originate from this infectious disease.

The nature of the pathologic process which I have postulated as the cause of the sensory loss related in my cases is not necessarily destruction of tissue, but a toxic action on the posterior columns, and very likely in some instances due to edema and capillary hemorrhages which usually clear up.

ABSTRACT OF DISCUSSION

Dr. W. W. Richardson, Mercer, Pa.: These cases were not uncommon in France. We had two cases of typical attacks of paraplegia following diphtheria at the base hospital center at Nantes. The patients were improving satisfactorily when they passed out of our hands. In discussing these cases with one of our consultant neurologists, he stated that they had had an epidemic of diphtheria at another base hospital center in France, and that they had there collected a series of twelve or fifteen of these cases. So, whereas we thought we had something quite rare in our two cases, they were really not very uncommon in France. The opinion was expressed by a prominent internist in France that if sufficient antitoxin had been used early in the diphtheria there would not subsequently have been so many nervous symptoms nor would they have been so severe.

Digitized by Google

A NEW PLURIGLANDULAR COMPENSATORY SYNDROME

WALTER TIMME, M.D.
Associate Physician, Neurological Institute
NEW YORK

From observation and clinical examination of many patients throughout the past six years at the Neurological Institute of New York City, I have come to the conclusion that the so-called types of endocrinopathies, such as status thymicolymphaticus, giantism, infantilism, acromegaly, and a host of unnamed others, are not static states, as one would be led to suppose from descriptions in the literature, but are simply cross-sections taken at intervals in a dynamic, progressive, and widespread disturbance of the internal glandular system. We had been struck at the Neurological Institute by the constant repetition of patients showing similar symptoms and similar physical signs which heretofore had been generally accredited to the asthenias accompanying neurasthenic states. The patients were usually of the late adolescent period, in the early twenties, and, passing over for the present the detailed symptoms which we will go into later on, the outstanding complaint overshadowing all others, was headache and extreme muscular fatigability. Accompanying these two suggestive conditions, there was an additional statement that they had been growing very rapidly for the past few years. We went deeply into their antecedent history and their family history, and were enabled thereby to find many points of resemblance in these various individuals. Finally, one patient, observed for six years, went through various stages to recovery at the age of 32 years. From

Digitized by Google

his early history we recognized symptoms that are presented by cases observed from time to time in the beginning stages of this syndrome, notably the fatigability, the headaches and the skeletal growth. In the past three years, so many cases of the kind have been observed that, from our past experience, we have been enabled to foretell, to a degree, the progress that such cases would undergo in the future. Many individuals admitted to the hospital for divers complaints of middle age revealed the final compensatory stages of the syndrome here to be presented. Being closely questioned, their antecedent history bore out in surprising detail the facts that our studied clinical types presented to us in various stages of the syndrome. They were completely compensated cases and their presence in the hospital was for some entirely adventitious cause. Our observation has netted us: (1) clinical types presented by single cases at various ages and stages of the syndrome; (2) progressive cases, observed over periods of from one to six years, showing the changing and probably compensatory nature of the disturbance; (3) completed cases in which the disturbance had come to a definite stop, in which the antecedent history bore out the close relationship to our isolated cases which were still in active progress; (4) uncompleted cases in which the condition, after going through the several preliminary stages, remained indefinitely progressive. To complete the chain it is necessary to observe at least one case from the beginning to the end of the disorder (a period varying from ten to twenty years), but as yet we have been unable to do this. Until this can be done we must fill in the gaps as best we can.

GENERAL DESCRIPTION

This new syndrome, pieced together as described above, may be generally stated to begin in youth some years before puberty, and go through its varying stages in about two decades. In its incipiency (first

Digitized by Google

stage) it presents largely the characteristics of the so-called status thymicolymphaticus, or status hypoplasticus of Bartels. There is complaint of muscular fatigability as a subjective sign, with frequent accompaniment of headache. Objectively the case presents usually, though not invariably (Fig. 6, Case 3, is an exception), an insufficient genital development, with perhaps inversion of sex type, with a penis that emerges from scrotal folds of labial type, or cryptorchism or both. In the female, the menses are usually delayed, the uterus and ovaries remain infantile and there is scarcity of pubic hair. Blood pressure is usually low, and blood sugar content is low. Eneuresis is common. There is usually present the white line of so-called suprarenal insufficiency of Sergent.

In the second stage, that beginning at puberty, we find a continuance of the muscular fatigability or even an increase. The genitals may remain backward or invert in development, the pubic hair is sparse and has the distribution of the opposite sex, the male showing a horizontal demarcation, while the female has the pyramid type in which the middle line to the umbilicus shows a hairy tendency. Axillary hair is absent and chin and lip show no signs of hair in the boy. Blood sugar is low, usually below 0.07 per cent., and blood pressure is below the normal. The white suprarenal line may be elicited; especially marked after fatiguing exercise. Roentgenograms of the skull usually show a sella turcica which is small or which may apparently be even enclosed by the clinoid processes. This is an important point to determine, for the later progress of the disorder presumabbly depends on the capacity of the pituitary gland to become hyperplasic. The size of the sella turcica, therefore, plays a determining rôle in the production of the later symptoms. The possible excessive function of the pituitary later on dominates the picture and is conducive to compensatory cure. The thymus gland frequently is

Digitized by Google

seen in the roentgenogram as quite enlarged. In some cases of extreme fatigability, I have also seen pineal shadows. During the second stage, rapid growth in length begins to become manifest—not ordinary normal growth—but rapid in the extreme, so that 5 or 6 inches in height a year is frequent. With this growth, fatigability increases, and it is on this account that the case is first brought to our attention.

In the third stage, we begin to see the results of some of the compensatory activities. It is usually ushered in about the 20th year of life. Growth has continued until the patient is 6 feet high or over; his weakness, even though his musculature seems splendid, is his prominent symptom. He shaves rarely or never. Pubic and axillary hair remain as before. Now he begins to notice an enlargement of his hands and feet, and a frontal headache or rather an intratemporal headache comes on. Blood pressure remains low (90 to 100 mm., systolic). Blood sugar usually remains low, but now frequently rises as compensation progresses. Our patient shows decided vagotonic symptoms. A roentgenogram of the skull at this stage, or during this stage, if the case progresses favorably, shows a sella turcica which, while small, may show erosion of the clinoids and a deepening of the cavity. This tendency of the pituitary to become hyperactive produces the headache (if the sella is contracted), the increase in blood sugar content, the growth of hands and feet, and a gradually rising blood pressure.

The fourth stage now comes on, from three to ten years later. This is the stage in which either complete compensation is produced or else in the untreated case there appear the varying and various attributes produced by an enlarged pituitary body engrafted on the earlier manifestations of a thymic state. That is, we have in the completely compensated case, features of acromegaly, although the blood pressure and blood sugar are normal and the headaches have gone. The

Digitized by Google

sella on roentgen-ray examination seems large. In the uncompensated cases we usually see a sella which is still small and perhaps bridged, with headaches of increasing severity, perhaps attacks of petit or grand mal, dependent on the disturbance of pituitary function, mental torpor, increase of weight with constantly increasing fatigue and a final lethal termination in intercurrent disease.

A brief review of the characteristic symptoms and findings in the different stages is here succinctly put forth:

First Stage.—The bony structure shows various endocrine anomalies and defects; disproportion of various skeletal units; teeth late and usually characteristically anomalous, so that the lateral incisors are very small, the central incisors disproportionately large, the canines of the type of incisors with a cutting edge instead of a tearing point; epiphyses slow in joining shafts of bones; hyperextension of joints; hair growth late and sparse, distribution of invert type; cramps in muscles; tendency to hemophilia and spasmophilia; enlarged thymus; maxillary torus; tonsils large and adenoids present; low blood pressure; low sugar content of blood; epistaxis; cyanosis of extremities; fatigability; small sella turcica; enuresis; low carbon dioxid coefficient of blood.

Second Stage.—At about the age of puberty rapid skeletal growth begins; late menses and small or infantile uterus; great fatigability with all evidences of low suprarenal supply; low blood sugar; low carbon dioxid combining power of the blood; white line of suprarenal insufficiency; pubic hair of invert type; lack of hair on face and chin and axilla; smooth soft skin of child; genitals of invert type or else retarded; vagotonia; symptoms of hyperacidity and gastric ulcer; enuresis; undue length of long bones; low blood pressure; little stamina.

Digitized by Google

Third Stage.—Twentieth to 30th year; beginning giantism; headaches, pituitary in character; drowsiness; acromegalic beginnings or other pituitary stigmata; fatigability may continue or improve; mental confusion and hebetude; epileptiform attacks—uncinate in type; sella turcica enlarges or else erosion of sella or clinoids takes place; blood sugar gradually increases if a cure is established. In uncompensated cases mental symptoms, moral and intellectual deficiencies and delinquencies arise.

Fourth Stage.—Either complete compensation, so that the patient may live comfortably within limits of exertion, or else progress to end of life as a pituitary case. The various external manifestations of the pituitary disturbance remain even if the physiologic cure is complete. Blood pressure rises; headaches cease; fatigability vanishes.

SYMPTOMATOLOGY

The bony structure in the first and second stages usually shows anomalies in proportionate skeletal growth; namely, legs too long for thorax or vice versa. (My scale for this determination is a fraction with the numerator as the distance from the sternoclavicular junction to the anterior superior spine of the ilium of the same side; and the denominator the distance from the anterior superior spine to the external malleolus. Normally, the fraction is one-half; a larger one meaning too large a torso, while less than one-half represents too long a leg. I call this ratio the torso-leg ratio.) The joints usually are hyperextensible and frequently the ligaments are so relaxed that dislocations are easily produced. The extremities can be thrown about like flails. While the teeth are usually delayed in their appearance, they also show certain characteristics. The lateral incisors, especially in girls, are frequently greatly underdeveloped. The canines, likewise, are either underdeveloped or else

Digitized by Google

take on the flat appearance of incisors, losing their fanglike appearance. With the cyanosis of the extremities we occasionally get a pustular-like eruption about the nails.

The symptoms during the second stage may need some elucidation. The so-called "white line" of suprarenal insufficiency was first described by Sergent. It has not been proved to be due to the deficiency, but in my experience it invariably accompanies low blood pressures, and may be made to disappear very quickly after a hypodermatic injection of epinephrin. Previous emotional disturbance, even so slight as that produced by standing before a camera to be photographed, will make its appearance impossible to obtain. It is obtained best by having the patient lie quietly in bed for a short while and then stroking the skin, preferably of the abdomen or thigh, lightly, with the palmar surface of the index finger. In 10 to 20 seconds, there will be a blanching of the skin thus stimulated. An article by Sergent gives the explanation of this phenomenon.[1] The low carbon dioxid combining power of the blood plasma diminishes the so-called "buffer" property of the blood and leads to acidosis on slight provocation. The smooth soft skin of these patients, even in the third decade of life, with little or no secondary hair on the face, a faint suspicion of lanugo on the lip and chin, and a "peaches and cream" complexion, frequently stamp these cases at sight. The headaches, produced as will be discussed in the pathogenesis, are of a specific type. They are invariably stated to be between the temples, the patient indicating the locality by putting one index finger on each temple, directed mesially. We have come to call them "pituitary headaches." The vagotonia present in many of the cases frequently takes on the character of hyperacidity of the gastric juice with frequently symptoms of gastric ulcer, spastic constipation and eosinophilia.

1. Sergent: Endocrinology **1**: 18 (Jan.) 1917.

Digitized by Google

In the third stage, the symptoms of great interest are the mental ones. In many years' observation of pituitary disorders, we have been frequently struck with the mental quips of the hypopituitaric. He exhibits lack of inhibition of the emotions, becomes highly excitable on little cause, alternating with sluggishness; frequently has phobias and compulsions (one case was a true kleptomaniac); shows frequently moral and sexual obliquities; and exhibition of pituitary feeding often modifies these characteristics. The symptoms of the uncompensated cases usually merge into those of a frankly dyspituitary syndrome. Blood sugar disturbances, intense fatigability, periodic headaches, temperamental unfitness and drowsiness are among the prominent symptoms.

ETIOLOGY

In practically all our cases there have been family histories of importance as regards endocrinopathies. Frequently, parents or grandparents have shown such disturbances as diabetes, goitre or acromegaly. A very common complaint is giantism. Collateral branches, too, show similar disturbances. Thus, W. W. (Fig. 10), the fully compensated case, has four cousins all afflicted with exophthalmic goiter. There appeared in our cases no particular antecedent disabling disease or injury. One patient, now in the second stage, had two brothers, both of whom died in youth suddenly, after exertion, from some unknown cause—possibly a so-called thymic death. Periodic headaches are also distinguishing marks, etiologically. Menstrual disturbances of all kinds are encountered, especially the late-appearing type with lack of periodicity.

PATHOGENESIS

During the first stage, we see a clinical picture which is dominated by the characteristics of the status hypoplasticus of Bartels. The anomalies have been variously credited to hypofunction of the individual

Digitized by Google

endocrine glands, excepting the thymus, which is supposedly hyperactive. Thus, Tandler and Gross and Tandler have described many of the features of such a condition due to deficiency of the gonads. And yet, in direct contradiction to their view that gonadal deficiency produces growth in height with late joining of the epiphyses, I have seen patients whom at the age of 18, with no menstrual flow yet established, the sexual apparatus quite infantile, the roentgenogram of the long bones showed the epiphyses almost united, whereas the height of the patients was under five feet. Wiesel, Schur, and Schmorls and Ingiers have given both clinical descriptions and histologic and pathologic findings in such hypoplastic conditions referable to underactive or inhibited suprarenal glands. Many observers have described the smallness of the sella turcica. My own observations agree with their descriptions. All of our cases show the smallness of the sella turcica in the early stages and in addition many of them have the bridging over by the clinoid processes, as becomes evident on roentgen-ray examination. With these deficiencies of glandular structure and their diminished physiologic activity ab initio, the organism would of necessity come to early grief if some corrective were not forthcoming. Many patients do succumb early. Undue exercise, sudden excitement, narcosis, are all critical moments for such organizations, many of which cannot survive them. After puberty should have been reached (the second stage) the deficiency of the gonadal inhibition to growth (Tandler and Gross) is claimed to be responsible for the extreme height rapidly reached in our cases. One of our patients (Private B.), however, rather opposes this theory in that the gonadal system early became hyperplasic and still the growth in body took place. This excessive genital development may be due in his case to an early involution of the pineal gland, for in the roentgenogram calcification of the pineal is seen. (Some authors

Digitized by Google

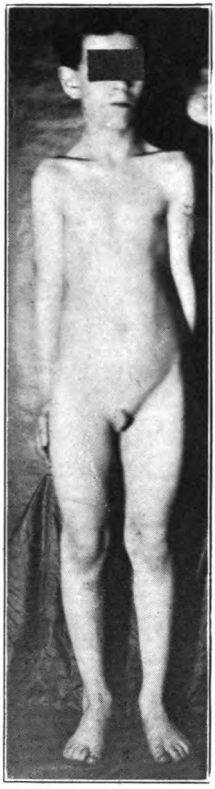

Fig. 1 (Case 1).—Abnormal length of thorax compared with legs; thighs especially short compared with lower leg; small genitals; large thymus and enclosed sella turcica.

Digitized by Google

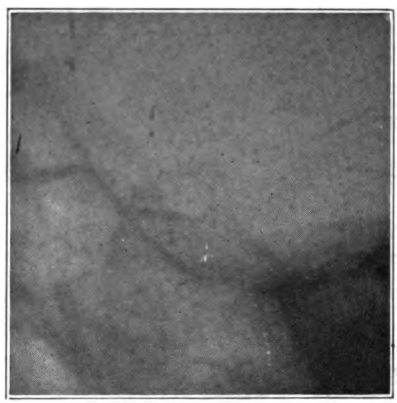

Fig. 2 (Case 1).—Sella turcica small and entirely enclosed and "roofed in."

Digitized by Google

Fig. 3 (Case 1).—Thymic enlargement.

Digitized by Google

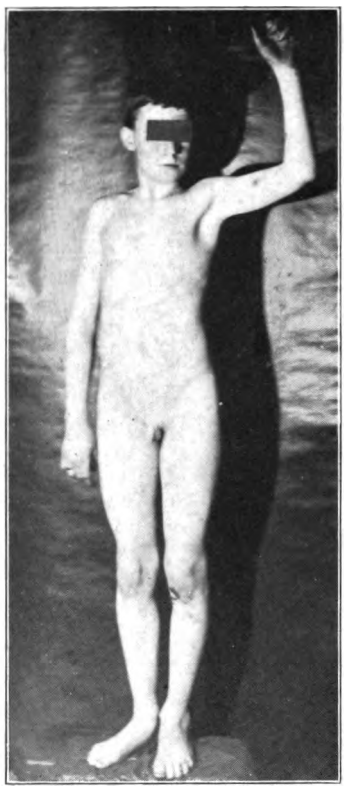

Fig. 4 (Case 2).—Age of patient 13½; hypoplasia of genitals and a scrotal fold surrounding base of penis; absence of hair; bruise on raised arm from slight pressure.

Digitized by Google

Fig. 5 (Case 2).—Pituitary fossa shut in extremely by large clinoids.

Digitized by Google

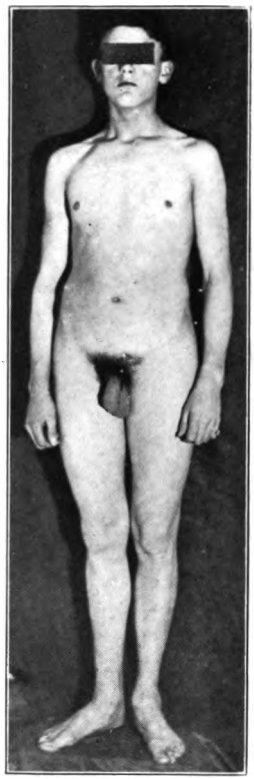

Fig. 6 (Case 3).—Feminine distribution of pubic hair; large genitals; height 6 feet 1 inch; no hair on face, well-proportioned general appearance.

Digitized by Google

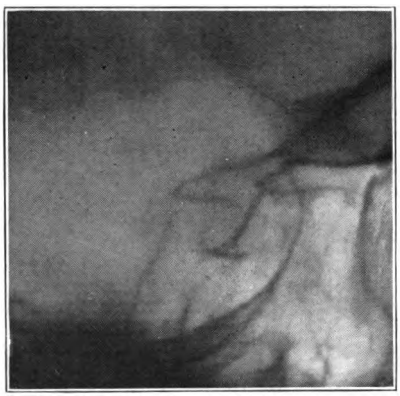

Fig. 7 (Case 3).—Complete closing in of pituitary fossa.

Digitized by Google

Fig. 8 (Case 4).—Height 6 feet 3½ inches; feminine pubic hair; no hair on face; musculature seems flabby; feminine attributes.

Digitized by Google

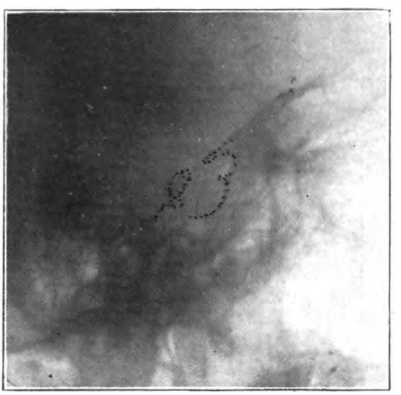

Fig. 9 (Case 4).—Sella turcica with anterior cavity eroded and a deepening of posterior fossa.

Digitized by Google

Fig. 10 (Case 5).—Height 6 feet 4½ inches; fully compensated case; great length of leg compared with length of thorax, feminine waist; feminine pubic hair; acromegalic hands; no hair on face.

Digitized by Google

Fig. 11 (Case 6).—Uncompensated case; pituitary fossa completely enclosed, as confirmed by stereoscopic plates; age of patient 22; height 6 feet.

Fig. 12 (Case 7).—Uncompensated case; age of patient 35; height 6 feet 1 inch; sella turcica completely shut in and extremely small, as confirmed by stereoscopic plates.

Digitized by Google

Digitized by Google

hold that the overactive thymus, with disturbed calcium metabolism, is the cause of such "thymic giantism.") The deficient suprarenal-chromaffin system is to be credited with the great fatigability, the low blood sugar content, the low blood pressure and the white line.

Now comes the third stage, the all-important one. It is in this period that the outcome of the syndrome is determined. In our judgment it is the pituitary gland that is here the critical factor. As we have seen, it is invariably enclosed in a small sella turcica and possibly even hemmed in by the clinoids. Among its functions we have, as all important, a blood pressor principle and a sugar mobilization factor. Both of these are deficient in our patient. If the pituitary possibly could become hyperplasic and hyperactive, with an intensification of these important properties, compensation might be accomplished. Such tendency to hyperplasia in a small cavity would of necessity through pressure produce headaches—an invariable symptom in the third stage of the compensated cases. And such headache would continue until the enlarged gland through erosion of its bony capsule or through pushing apart the clinoids made sufficient room for itself. As will be seen, these headaches continued for two to three years in some of our cases. Synchronously with these headaches, other incidental features of an enlarged pituitary gland became manifest: (*a*) acromegaly, lasting until the headaches ceased and the process then likewise ceasing; (*b*) a higher blood sugar content; (*c*) a higher blood pressure, and (*d*) a diminished sugar tolerance. To make this view of the nature of the process of compensation more tenable, many of the sellae turcicae of our patients in the second and third stages show erosion of anterior or posterior clinoid processes; and in the final stage, an enlarged sella with practically no clinoid processes left at all. In the cases in which no compensation was effected; namely, in which fatigability and the other symptoms remained and progressed, the sella

Digitized by Google

showed no enlargement (notably that of T. R., Fig. 12). In these cases, we had headaches, periodic in type, adiposity, mental and moral deficiencies, petit mal and other manifestations. Curiously enough, in all our cases, the feeding of the pituitary gland in fairly large quantity, disposed of many and at times of all of these symptoms. But if the feeding should be diminished or stopped, the symptoms reappeared. It seemed analogous to thyroid feeding in myxedema. One case, which gave a typical early history and seems uncompensated today at the age of 44, still shows the very small sella turcica with a clinical picture of abnormal bony structure much resembling Paget's disease. On pituitary feeding, this case is improving markedly in its features of fatigability, headaches and heaviness of extremities. It is too early to state whether in her case the cancellous condition of the bones will be restored.

Finally, the fourth stage is ushered in by a gradual cessation of the fatigue, amelioration of the headaches, restoration of the normal blood pressure and normal sugar content of the blood. But the adventitious signs of the disturbance of the pituitary gland remain. Thus the fully compensated cases may show acromegaly more or less marked; *and this acromegaly is not to be taken as a diseased condition needing treatment, but simply as the hallmark of a process that has come to a stop—a self-curative process.* It is analogous to the hypertrophied heart become so through the deficiency of the cardiac valves and making up for such deficiency by its enlargement. And that condition likewise, per se, needs no treatment. A case that presents acromegalic features, therefore, need not necessarily be a case that calls for therapeutic intervention. It may well be a "finished" case. W. W., Figure 10, is a good representation of this type. These "finished" cases must always, however, live within certain limits of exertion and stress. The cases that in the fourth stage do not

Digitized by Google

spontaneously go to full compensation are those in which we either find a sella turcica that did not enlarge (perhaps because there was no spontaneous effort of the pituitary to become hyperactive) or in which an enlargement of the sella did take place and the pituitary even in its hyperactive condition was not sufficient to compensate. These uncompensated cases go right on with progressive symptoms of fatigability, asthenia, headaches and so forth, making of them easy prey to intercurrent affections.

TREATMENT

The treatment of these cases in any stage is extremely satisfactory. The great point to remember is the probable nature of the process of compensation which the organism is endeavoring to carry out. That would make one believe that suprarenal gland therapy is indicated throughout on account of the patent deficiency of this organ in these cases. And yet in our hands its administration is disappointing. The whole gland perhaps has given better results than epinephrin, although the latter, either hypodermically or (even against the dictum of the physiologist that it is inert when administered per os) by mouth in larger doses is good to tide over exceptionally bad days of fatigue and exhaustion. But the prime agent—almost a specific one—is pituitary gland in some one of its varied forms. Whole gland feeding in fairly large doses, from 2 to 3 grains three times a day, may be given in appropriate cases. But usually the dosage should be much smaller, and given at greater intervals; one half or one fourth grain every second or third day has given success in several patients. Occasionally, pituitary extract hypodermically, from 0.5 to 1 c.c. per day or on alternate days, for one or at most two weeks at a time, is excellent as supplementing the feeding of pituitary gland. Occasionally in cases with pronounced genital delay, anterior lobe pituitary gland gives fair results. In those cases with vagotonic symptoms, hyperacidity and

Digitized by Google

conditions resembling gastric ulcer, atropin in doses to physiologic tolerance is indicated, and gives results. But the pituitary feeding in itself, alone, produces highly satisfactory improvement in almost every case. Under its use, the headaches disappear, the fatigability diminishes, the blood pressure and blood sugar content increase, and the case goes on to cure. Gradually the pituitary feeding can be diminished and finally discontinued. In the older cases, in which the sella persists in remaining small (T. R., Fig. 12) and (J. S., Fig. 11) constant feeding would seem to be necessary; at all events, the patients relapse as soon as treatment is stopped. Indeed, the patients themselves reach that point of accuracy of judgment in feeding the gland to themselves that they can determine the size and frequency of the dosage necessary to maintain them comfortably.

The case histories are cited as a few among many that show characteristics of the different stages of the syndrome. Only facts having direct bearing are introduced into the case histories.

CASE 1 (Fig. 1)—*History.*—J. M., aged 10, was mentally backward. There was no hair on the body anywhere. The genitals were undeveloped. The thorax was elongated. Spasms in the muscles occurred. Great fatigability was present. He was a bed wetter. The joints were hyperextensible. He was subject to nose bleeds. A roentgenogram of chest shows thymus (Fig. 3). The skull shows a small sella turcica, entirely bridged over (Fig. 2). Blood pressure is 85. The white suprarenal line is present.

Comment.—This case is presented simply to show the type from which arise the cases presenting the later features of the syndrome and hence may be classed under Stage 1.

CASE 2—*History.*—F. J., aged 13½ years, height, 61½ inches, was brought to the Neurological Institute on account of his predilection for lying and further for his rapid fatigability. He had been discharged from several schools on account of his incorrigibility. The climax was reached when he appeared as a material witness in a murder trial, his evidence being of the greatest importance in the conviction of the accused. Apart from these facts, he also complained of spasms in the muscles, especially of the calves, which

Digitized by Google

occasionally awakened him by the pain caused thereby. He had frequently also had nocturnal enuresis. On stripping him (Fig. 4) the examination revealed a body perfectly hairless, no trace of pubic or axillary hair being evident. The genitals were small—infantile in fact—but both testicles were descended and the scrotum surrounded the penis like a labial fold. On eliciting the cremasteric reflex, however, the testicles were drawn up out of the scrotal sac into the inguinal canal where they could not be palpated. He bruises very easily, the dark patch on the left raised arm being produced by the simple pressure of the examiner's thumb in raising the arm. Hands and feet are somewhat larger than they should be. Stroking the skin of the abdomen produced a marked white line of reaction, which persisted for a rather long time. His blood pressure was low, 80 mm. Mentally he was well up to his chronological age according to the Binet-Simon scale. Being closely questioned, he admitted that he had frequent headaches referable to a point midway between the temples and deep-seated.

The neurologic examination was negative. Laboratory examination showed the sugar content of the blood to be 0.07 per cent. Otherwise, blood and urine were negative and the Wassermann test was negative. Roentgen-ray examination: The sella turcica (Fig. 5) was small and encroached on by large clinoids. The upper thorax showed a thymus shadow.

Comment.—This case is presumably a type of the beginning of the second stage of the syndrome. It presents many of the well marked features of a status hypoplasticus—small sella, enlarged thymus, low blood pressure, low blood sugar, marked fatigibility, spasmophiliac and hemophiliac attributes in addition to the obvious externals. The mental attributes are those which we frequently see in small and enclosed sellae turcicae. This case is presented simply as a living example of the early beginnings of the second stage of the syndrome.

CASE 3 (Fig. 6)—*History.*—Private B., aged 20, height 6 feet 1 inch, was sent to the institute by Captain Reed, stationed at Fort Wood, to determine whether or not the man was a malingerer. His one complaint was that of excessive fatigability after moderate exertion. As a result he could not perform the military duties required of him. His musculature and his whole bearing and appearance when stripped were that of a powerfully and symmetrically built young giant, and seemed to belie his statement of rapid fatigue. His early history could not be obtained with any degree of reliability for, coming from a mountainous district of the South, he was extremely uncommunicative. In early adolescence he was a rover, traveling over many states in divers occupations, but never steadily at any one. He did say that for the past three years he had been rapidly and steadily growing in height, that this growth was still continuing and with it his fatigability

Digitized by Google

was increasing. Of note was the fact that he had practically no hair on his lip or chin, no hair in the axillae, pubic hair of the feminine type and rather largely developed genitals. Stroking the skin produced a white persisting reaction. His blood pressure was between 95 and 100 mm., systolic; 80 mm., diastolic. Blood sugar was 0.062 per cent. Roentgen-ray examination showed a sella turcica (Fig. 7) that was extremely small, with thickened anterior and posterior clinoid processes completely roofing in the cavity. A shadow in the pineal region also was evident. The thorax showed a thymic shadow. The neurologic examination was negative. The viscera appeared normal.

Comment.—This young man is going through the transition period from the second stage to the third. His low blood pressure, low blood sugar percentage (extreme normal low limit should be at least 0.075 per cent., white skin reaction line, all go to prove his statement of fatigability. In addition he shows a crowded pituitary fossa and a pineal shadow. We have found in the past two years in a large percentage of muscular dystrophies pineal shadows; and in extreme cases of muscle fatigue in the adolescent, short of dystrophy, there were shadows in the pineal region. This parallelism between the myasthenic types and early involution of the pineal gland will be the subject of a future paper. This feature of the case is an example of many similar ones. But presumably early pineal involution also produced his enlarged genitals. He is undergoing the rapid growth incident to the second stage of the syndrome in spite of the fact of the enlarged genitals. Some authors (especially Tandler and Gross) have maintained that the hypoplasia of the genitals accounts for the continuance of the skeletal growth. That is certainly disproved here as a universality. On feeding of pituitary gland, this youth improved sufficiently to go back to army life. After the lapse of a few months, he discontinued this feeding and shortly thereafter returned with his old complaint of fatigability. He is again improving on treatment. No white line can now be elicited, and his blood pressure, is usually at 120 mm.

Case 4 (Fig. 8)—*History.*—G. E. R., aged 22 years, height 6 feet 3½ inches, single, stenographer, came to the Neurological Institute complaining of loss of memory and lack of sleep, of feeling stuperous and of being extremely fatigued. He has headaches intratemporal in location. He is not able to do much manual work on this account and hence he took a position as stenographer.. He is exempt from military duty, being the sole support of his mother.

While a child, he was never strong; he took a long time to develop and on account of his ways (feminine) was always known as "sissy" to his playmates. He realized this appellation to be more or less just. He would easily cry if deprived

Digitized by Google

of his way and he still does so. He always was a bed-wetter, and this weakness persisted to his 19th year. Realizing his feminine attributes, he endeavored to compensate for them by indulging in the most manly of sports—boxing and football. But he was too slow to amount to anything here. He gave up this conflict and sank to his own recognized level—became a stenographer. He began to get headaches at 17 years of age, at which time his excessive growth started.

Examination.—The patient was a man powerfully set-up, 6 feet 3½ inches high and proportionately built (Fig. 8). There was no hair on the face or the body except in the pubic region, where it was of feminine distribution. The scrotum also was divided above the penis into typical labial folds. He shaves once in two weeks. His appearance is really pugnacious, but is not borne out by his mental attitude. He would cry when questioned about the simplest difficulties. His blood pressure was 100 mm., systolic. He had the white line reaction of the skin, and his blood sugar was low. The roentgenogram of the skull revealed a sella in which the erosion of the anterior clinoid is evident and an excavation of the posterior portion of the floor (Fig. 9). The roentgenogram of the thorax shows a possible enlargement and persistence of the thymus. The neurologic examination was negative.

Comment.—The early history was that of a thymus state. The further progress, especially the headache concomitant with the growth, was suggestive of pituitary enlargement. This was partly borne out by the picture of the sella. The headaches are now much less prominent than they were, but his fatigability is marked. His growth seems to have ceased. This case belongs to the third stage, with giantism gradually reaching its acme, with the stuporous and drowsy mental condition of a dyspituitaric still present, great fatigability, low blood pressure and low blood sugar content. The enlargement of the sella turcica, however, gives promise of a gradual efficient compensation taking place. Feeding of whole gland pituitary extract within two weeks measurably improved all his symptoms. It is still being continued.

Case 5—*History.*—W. W., aged 32, a machinist, married, several children, height 6 feet 4½ inches, had acromegaly in a slight degree. This case has been under our observation since 1912. At first the complaint was one of utter exhaustion to the exclusion of all minor ills. He could not stand at his work, he could hardly walk without becoming exhausted. At 19 he had severe headaches referred to the intratemporal region, which persisted in spite of all measures. These headaches he described as "crushing." At the same time his hands enlarged while under our observation. The fatigability was so great that he would have to take to his bed and remain there. His past history was typical of the thymic. Nose

Digitized by Google

bleeds, general weakness, enuresis, overextensibility of the joints. His family history was a typically endocrine one. On his mother's side four members of direct and collateral branches had goiter, two with exophthalmos. His father never shaved until the age of 28. Puberty in this patient was reached at 19. Hairy growth in the axilla and on the face remained absent. To this day he has never shaved. During the period of intense headaches and growth, he had various vagotonic symptoms: hyperacidity, excessive perspiration, precordial distress, nausea and vomiting, marked pallor, lack of libido. His gastric distress became so acute that at another hospital operation for gastric ulcer was advised. At this time a roentgenogram of the skull was taken and showed a deep sella turcica, with practically no anterior clinoids present at all and erosion at the edges, with the posterior clinoids seemingly pushed backward and upward into the skull cavity. His photograph (Fig. 10) has some interesting points. The extreme length of leg as compared with the thorax is noteworthy. The feminine distribution of pubic hair and a feminine waist are present. His extremely large hands and generally deficient musculature attract notice. This photograph was taken about a year ago when he was just completing his cure. Today he has gained 30 pounds in weight and is feeling practically well; is working constantly as machinist and supporting his family, consisting of wife and two children. During the last three years, his blood pressure had gradually risen to 130 mm., systolic, from an initial 90 to 100 systolic; his gastric symptoms have abated; the white line of suprarenal deficiency is disappearing; his fatigability has passed; libido has returned, and he is to all purposes cured of his malady. His fatigability and headaches in the past three years could always be improved with the administration of whole gland pituitary extract and gradually it became possible to diminish the dose and allow the compensation to proceed unaided. He has now been without treatment for practically six months. His seems to be a fully compensated case.

Comment.—The history and progress of this case is typical of all cases passing through the complete syndrome and is presented as a type of which I have now seen at least ten examples. It must not be forgotten that he now shows giantism plus acromegaly. That does not mean that his clinical picture indicates treatment—far from it. He represents a finished case. The giantism and acromegaly are incidents merely of the compensatory process. They themselves do not necessarily indicate present disease. This is a point well worth remembering. Many cases showing endocrinopathic features come under our notice and it should be our first effort to determine whether or not they represent processes that have come to a stop through compensatory efforts

Digitized by Google

of which they are the indices. Under such conditions of course no treatment is called for. An interesting case of this nature was seen at Mount Sinai Hospital, New York, in the service of Dr. Goldenberg, through the kindness of Dr. I. Strauss. A woman, 53 years of age, with typical acromegaly —hands, feet and skull abnormalities—was admitted for some minor ailment. Her history showed that she began to menstruate at 19 years; had intense headaches at 25 years, which for three years resisted all attempts to alleviate, and during the persistence of which she began to grow acromegalic. When the headache spontaneously ceased, the abnormal growth ceased, and she passed an uneventful life to the time of the present slight ailment. Her childhood and adolescence were like those described in the first two stages of this syndrome, fatigability being the prominent symptom. Her acromegaly, then, was completed at about the twenty-ninth year; did not further increase, and she remained well for twenty-three years longer. This was also a "finished" case as far as the syndrome of thymus, suprarenal and pituitary was concerned. This case might be many times duplicated.

UNCOMPENSATED CASES

I desire to give rapidly only the salient features of several uncompensated cases belonging to the syndrome.

CASE 6.—Miss J. S., height 6 feet, large hands and feet, referred by Dr. Robert T. Morris, was suffering from extreme fatigability, headaches and vagotonia. There had been rapid growth the previous two years, with a past history of status thymicolymphaticus. She came for relief from her headaches and fatigability. Blood pressure was 95 mm., systolic; blood sugar, 0.065 per cent.; fatigue was so great that she had to remain prone the greater part of the day. There was an intense white line. The sella turcica (Fig. 11) showed a roofing in the clinoids. Pituitary feeding gave gratifying results, but must be continuously carried out.

CASE 7.—T. R., aged 35, height 6 feet 1 inch, obese (220 lbs.), suffered from early fatigability. There was evidence of late puberty. Pubic hair was feminine. He was unmoral. He had intense intratemporal headaches. Although wealthy, and of excellent family, he would commit sexual and other excesses. He was frequently in jail. Fatigability and headaches persisted. With the beginning of headaches growth began. Blood pressure was 95 mm. The sella turcica was small and closed in by the clinoids (Fig. 12); polyuria was present. Pituitary feeding helped not only his headaches and fatigability, but, strangely and unexpectedly, his moral sense

Digitized by Google

became elevated and his excesses are no longer indulged in. But the pituitary feeding must be constantly kept up. A few weeks of discontinuance leads rapidly to the old condition. He has been now practically free of his troubles for two years.

CONCLUSION

The syndrome of thymus-suprarenal-pituitary combination is one frequently encountered and its various stages are easy of recognition. The main characteristics of fatigability, low pressure, headache and growth are invariable components of the syndrome and depend on maladjustments of endocrine interactivity. Stabilization of the balance may be spontaneously produced provided the sella turcica may be made to accommodate a hyperactive hyperplastic pituitary gland. This is done presumably by erosion of the bony capsule of the gland. In cases of inability of such enlargement of the sella, the syndrome persists, but the symptoms may be alleviated by the feeding of pituitary extract continuously. In the course of the syndrome other glands may be brought into the complex and alter the picture somewhat, but these are vagaries and seemingly have no great determining effect on the course of events. Once recognized in any of the early stages, the further general progress of these cases can be prognosticated with a great degree of accuracy; and intervention, if necessary, can be undertaken with a large degree of success in the amelioration of the distressing symptoms.

2 West Sixty-Seventh Street.

ABSTRACT OF DISCUSSION

Dr. Tom A. Williams, Washington, D. C.: There are cases in which some symptoms are absent. I have now under observation a young girl whose blood pressure so far from being low is 133. The most striking feature of her case is the character of the biochemical changes. In 1911 I reported the case of a child, 11 years of age, whom we fed on pituitary for six months. That child was a pathologic liar, which I interpreted as a defense reaction to the symptoms produced by the hypopituitarism. That was eight years ago and she has entirely recovered. Perhaps in some of these cases of

Digitized by Google

Dr. Timme's we may be able to dispense with pituitary extract eventually after a certain degree of compensation has been established. Where there is much pain accompanying the intracranial pressure it might be desirable to try to reduce the dimensions of the hypophysis by the roentgen ray. I have succeeded in at least three of my cases of pituitary disorders.

Dr. Albert E. Sterne, Indianapolis: We should keep in mind one essential point and that is the antagonism between the anterior lobe and the posterior lobe, not forgetting the middle lobe, and accent the necessity of differentiating the cases into preadolescent and postadolescent types. It makes a great deal of difference as to the time at which the physician sees the particular case. Both in hypotypes and hypertypes certain rather definite hormonic physical signs are developed, but these are apt to vary, depending on whether abnormal pituitary function had been in play before or after adolescence. The mere presence of these bodily signs is not necessarily conclusive evidence of the state of hypophyseal activity at the time the doctor observes and examines the patient. In hypopituitarism the preadolescent hormonic somatic characteristics may still be apparent, but there may, and often does, occur a relative compensation which lessens the deficient pituitary function and the somatic signs begin to modify somewhat but not usually to disappear. Many of these patients look like brothers and sisters but are not blood related at all. Dr. Timme did not mention all of the peculiarities that present themselves, such as the appearance of the teeth, for instance, in the hypopituitaries. Again, the disproportion in the long bones is evidence of posterior lobe overfunction, whereas in anterior lobe deficiency, we get the fatty deposit, usually about the trunk, not on the arms and legs, which, in proportion, are symmetrical and small. These are important points to take note of, because the question of therapy comes into play. In my experience we can feed many of these patients on the comparatively harmless anterior lobe and gain comparatively slight, if any, results. In the use of the posterior lobe liquid, it is necessary, in order to accomplish the best effects, to give hypodermic or deep intramuscular injections and it is very important to regulate, individually, the dose used. It is my custom to give 5 drops first and, if that gives no results, to raise the dose. We usually have found 15 drops to be an average dose, effective without arousing a constitutional reaction. Feeding by mouth has proven comparatively inefficacious, whereas the deep intramuscular or subcutaneous injection of the pituitary liquid has proven efficacious in a number of instances.

Dr. Walter Timme, New York: As far as Dr. Williams' comments are concerned, of course we do not meet with cases that do not show all the characteristics of the syndrome and it is sometimes difficult to know if they fit exactly in it. In

Digitized by Google

medical science there is no one syndrome that does not dovetail into another. If you get a case without giantism, no headache, normal blood pressure combined in one patient, I would say that case fits somewhere else. Regarding Dr. Sterne's remarks, I did not touch on the minutiae of examination or therapy. The time was insufficient to more than indicate the salient features of such a syndrome as this. This is an endeavor to put together apparantly various types and the reasons therefor, and the subject of therapy could hardly be touched. For a similar reason I said nothing about teeth, hyperextension of joints, long bones, etc. As far as the therapy is concerned, however, Dr. Sterne's statement as to the harmlessness of the character of the anterior lobe requires a reply. It is far from harmless. It will produce growth in organs in which you do not desire it. In cancer in mice the growth has been tremendously accelerated by this substance. Hence therapy with the anterior lobe must be undertaken with caution. The posterior lobe is used only temporarily because its continuous use leads to disturbance of sugar metabolism and marked asthenia. Our process of therapy is the feeding of the fresh whole gland.

BIBLIOGRAPHY

Maranon: Insuffisance plurigland. (Atrophic testiculaire acromégalique), Rev. clin. de Madrid, 1909, p. 330.

Marie, Pierre: Persistance du thymus chez n acromégalique, Soc. méd. des Hôpit. **2**: 17, 1893.

Wiesel: Zur Pathologie d. Chromaffin Systems, Virchows Arch. f. path. Anat. **176**: 103, 1904.

Tandler: Ueber den Einfluss d. innersekret. Anteile der Geschlechtsdrüsen auf die äussere Erscheinung des Menschen, Wien. klin. Wchnschr. **23**: 459, 1910.

Tandler und Gross: Ueber den Einfluss der Kastration a. d. Organismus, u. s. w. Arch. f. d. Entwicklungsmechanik **27**: 35, 1909.

Cushing: The Pituitary Body and its Disorders. Especially Case 32 (giant Turner). This case seems to fit into the above described syndrome almost perfectly: lack of hair, small genitals; low temperature, minute suprarenals, intense muscular fatigability, growth beginning to be excessive at age of 15 accompanied by intense headaches. Cushing himself states that "the patient is not an acromegalic giant" in the true sense (p. 168).

Bartel: Status thymico-lymphaticus and status hypoplasticus, Vienna, 1912.

Lucian, M., and Parisot, J.: Contribution à l'étude des fonctions du thymus, arch. de méd. expér. et d'anat. path. **22**: 98, 1910.

Müller: Fall von Akromegalie, Osteomalacia, Tetanie und Struma, Wien. klin. Wchnschr. **22**: 76, 1909.

Ingiers und Schmorle: Ueber den Adrenalingehalt der Nebennieren, Deutsch. Arch. f. klin. Med. **104**: 125, 1911.

Schur und Wiesel: Ueber d. Verhalten d. Chromaffin. Gewebes bei d. Narkose, Wien klin. Wchnschr. **21**: 247, 1908.

Renon, Arthur Delille et Monier-Vinard: Syndrome polygland (thyreo-testiculaire-hypophys.) Soc. méd. des hôpit. **2**: 5, 1909.

Massalongo, Sull' acromegalia, Riforma med, **8**: 157, 1892.

Hoskins, R. G.: Relation of the Adrenal Glands to the Circulation of the Blood, Endocrinology **1**: 292, 1917.

Digitized by Google

INDEX

PAGE

Abbot, E. S.: discussion on psychiatric diagnosis 114
Amnestic fugue, account of; paper by E. E. Mayer..... 59
Discussion 81
Anemia, resemblance of sensory symptoms of post-diphtheritic ataxia to those seen in cord changes of severe anemia; paper by G. Wilson 177
Discussion 183
Angell, E. B.: discussion on pituitary in epilepsy..... 174
Ataxia, post-diphtheritic, resemblance of sensory symptoms of, to those seen in cord changes of severe anemia; paper by G. Wilson 177
Discussion 183
Bassoe, P.: paper on epidemic encephalitis 158
Discussion on same 168
Brain, congenital tumor of, and associated cerebral movements; paper by W. G. Spiller 149
Discussion 156
Byrne, J.: discussion on dual personality..... 81
Caro, H.: paper on neurologic sequelae following acute respiratory infections 127
Church, A.: paper on neurology of cervical ribs..... 17
Crafts, L. M.: paper on original test for pathologic great toe sign.. 116
Diagnosis, psychiatric, range of general practitioner in; paper by E. E. Southard 103
Discussion 114
Diphtheria, post-diphtheritic, resemblance of sensory symptoms of, to cord changes of anemia; paper by G. Wilson..... 177
Discussion 183
Drysdale, H. H.: paper on hysterical hemiplegia.....136
Discussion on same 147
Dual personality of C. A. M., the account of an amnestic fugue; paper by E. E. Mayer..... 59
Discussion 81
Encephalitis, epidemic, contribution to histopathology of; paper by P. Bassoe and G. B. Hassin..... 158
Discussion 168
Epilepsy, rôle of pituitary gland in; paper by B. R. Tucker.... 169
Discussion 173
Gardner, J. S. S.: paper on hysterical hemiplegia 136
Gordon, A.: discussion on acute respiratory infections..... 135
Discussion on hysterical hemiplegia..... 144
Grinker, J.: discussion on hysterical hemiplegia..... 147
Hassin, G. B.: paper on epidemic encephalitis 158
Hemiplegia, hysterical hemiplegia; paper by H. H. Drysdale and J. S. S. Gardner 136
Discussion 144
Hicks, W. H.: discussion on war hysteria 55
Hoppe, H. H.: discussion on pituitary in epilepsy 174
Discussion on tumor of brain 156
Hysteria, war, management of; paper by T. A. Williams..... 38
Discussion 54
Jones, W. A.: discussion on dual personality 82
Kennedy, F.: discussion on hysterical hemiplegia 146
Langdon, F. W.: discussion on tumor of pons variolii..... 126
Lindsay, W. S.: discussion on hysterical hemiplegia 146

Digitized by Google

PAGE

McGregor, R.: discussion on war hysteria 54
Mayer, E. E.: paper on dual personality 59
Discussion on same 84
Discussion on hysterical hemiplegia 147
Mayer, W. H.: discussion on acute respiratory infections..... 133
Mills, C. K.: paper on neurologic therapeutics 85
Moleen, G. A.: discussion on new test for toe sign..... 121
Discussion on pituitary in epilepsy 174
Neurologic sequelae following acute respiratory infections; paper by W. H. Robey and H. Caro 127
Discussion 133
Neuroses, traumatic neuroses in war and in peace; paper by T. H. Weisenburg 29
Discussion 54
Patrick, H. T.: discussion on hysterical hemiplegia..... 145
Pituitary gland in epilepsy; paper by B. R. Tucker..... 169
Discussion 173
Pluriglandular compensatory syndrome, new; paper by W. Timme 184
Discussion 202
Pollock, L. J.: discussion on hysterical hemiplegia 145
Discussion on war hysteria 56
Pons varolii, tumor of; paper by T. B. Throckmorton 123
Discussion 126
Psychiatric diagnosis, range of general practitioner in; paper by E. E. Southard 103
Discussion 114
Respiratory infections, neurologic sequelae following; paper by W. H. Robey and H. Caro 127
Discussion 133
Ribs, cervical, neurology of; paper by A. Church..... 17
Richardson, W. W.: discussion on post-diphtheritic ataxia and cord changes of severe anemia 183
Robey, W. H., Jr.: paper on neurologic sequelae following acute respiratory infections 127
Discussion on same 135
Rosewater, C. A.: discussion on acute respiratory infections..... 133
Discussion on war hysteria 55
Sands, I. J.: discussion on pituitary in epilepsy 175
Scalp, report of case of hysterical hemiplegia, resulting from shrapnel wound of scalp and presenting interesting clinical features; paper by H. H. Drysdale and J. S. S. Gardner..... 136
Discussion 144
Singer, H. D.: discussion on dual personality 81
Southard, E. E.: paper on general practitioner in psychiatric diagnosis 103
Spiller, W. G.: paper on congenital tumor of brain 149
Discussion on same 157
Sterne, A. E.: discussion on dual personality 82
Discussion on hysterical hemiplegia 144
Discussion on pituitary in epilepsy 175
Discussion on pluriglandular compensatory syndrome .. 203
Telangiectasis, congenital, of brain; paper by W. G. Spiller..... 149
Discussion 157
Therapeutics, neurologic, some experiences in; paper by C. K. Mills 85
Throckmorton, T. B.: paper on tumor of the pons varolii 123
Timme, W.: paper on pluriglandular compensatory syndrome.... 184
Discussion on same 203
Discussion on pituitary in epilepsy 173, 175
Toe, original test for pathologic great toe sign; paper by L. M. Crafts 116
Discussion 121

Digitized by Google

PAGE

Tucker, B. R.: paper on rôle of pituitary gland in epilepsy 169
Discussion on same 176
Tumor, congenital, of brain and associated cerebral movements; paper by W. G. Spiller 149
Discussion 156
Tumor of pons varolii, case of; paper by T. B. Throckmorton 123
Discussion 126
War hysteria, management of; paper by T. A. Williams..... 38
Discussion 54
Weinburg, M. H.: discussion on acute respiratory infections 134
Discussion on epidemic encephalitis 168
Weisenburg, T. H.: paper on traumatic neuroses in war and in peace 29
Discussion on same 57
Williams, T. A.: paper on management of war hysteria 38
Discussion on same 58
Discussion on dual personality 83
Discussion on hysterical hemiplegia 144
Discussion on pluriglandular compensatory syndrome 202
Wilson, G.: paper on sensory symptoms of post-diphtheritic ataxia 177

Digitized by Google

Digitized by Google

Digitized by Google

Digitized by Google

4P·C
614

Digitized by Google

Digitized by Google

48·C
614

Digitized by Google

Digitized by Google

48·C
614

Digitized by Google

Digitized by Google

48·C
614

Digitized by Google

3 2044 102 962 982